Complainant's
Exhibit No. 115

EXHIBITS

Deposition of:

GERALD KENNEDY

VOLUME I
EXHIBITS 1 - 24

DATE OF DEPOSITION:

JULY 31, 2002

IN THE CIRCUIT COURT OF WOOD COUNTY, WEST VIRGINIA

JACK W. LEACH, et al.,

Plaintiffs,

v.

CIVIL ACTION NO. 01-C-608
(Judge George W. Hill, Jr.)

E.I. DU PONT DE NEMOURS AND COMPANY, and LUBECK PUBLIC SERVICE DISTRICT,

Defendants.

AFFIDAVIT OF GERALD KENNEDY IN SUPPORT OF E.I. DUPONT DE NEMOURS AND COMPANY'S MEMORANDUM OF LAW IN OPPOSITION TO PLAINTIFFS' SECOND MOTION FOR SANCTIONS

STATE OF DELAWARE, COUNTY OF NEW CASTLE

GERALD KENNEDY, being duly sworn, deposes and says:

1. I have worked for defendant, E.I. Du Pont de Nemours and Company ("DuPont") from 1977 to the present. I work in DuPont's Haskell Laboratory for Health and Environmental Sciences. I am currently the Director of Applied Toxicology and Health.

2. On November 15, 2001, DuPont entered into a consent decree with the West Virginia Departments of Environmental Protection and Health & Human Resources to determine whether there has been any impact on human health or the environment from DuPont operations at the Washington Works facility in West Virginia.

EXHIBIT
i
Kennedy

3. I am part of the team called the C-8 Assessment of Toxicity ("CAT") Team established by the November 15, 2001 consent decree to assess the toxicity and risk to human health and the environment associated with exposure to C-8 releases from DuPont activities at the Washington Works facility.

4. From approximately January 2002 through May 2002, I received emails from Dr. Dee Ann Staats, a toxicologist with the West Virginia Department of the Environment ("WVDEP") who serves as chair of the CAT Team. I do not recall receiving a large number of emails from Dr. Staats. In general, these emails were of a scheduling nature requesting, for example, dates of availability for meetings, providing meeting logistics or explaining that a meeting needed to be rescheduled.

5. The toxicologists involved in the CAT Team met on May 6-7, 2002 in Cincinnati, Ohio to set human health screening levels for C-8. At some point, WVDEP had retained the services of Toxicology Excellence for Risk Assessment ("TERA"), an organization based in Cincinnati that has significant experience in toxicology/risk assessment issues. TERA, whose representatives were members of the CAT Team, provided a handout of information to be used at the May 6-7, 2002 meeting. Dr. Staats either directly or through TERA sent the handout to all CAT Team members prior to the meeting. I recall receiving the handout electronically, printing it out, and bringing it with me to the meeting along with some logistics information about how to get to the meeting.

6. At the May 6-7, 2002 CAT Team meeting, Dr. Staats designated an individual from TERA as the official notetaker for the meeting. During the two day meeting, the exercise of setting the screening levels required some arithmetic. On approximately two sheets of paper, I, from time to time during the meeting, wrote out numbers on the paper to check the arithmetic calculations of the Team. I was not the official person responsible for those calculations, but was simply double checking the math from time to time. Because there was an official notetaker charged with retaining information and there was nothing of substance to my "checking of arithmetic" notations, I did not retain the approximately two pieces of paper after the meeting. For the same reasons, I did not retain the complete TERA handout, but did keep three pages of it, some of which have my handwritten notations on them.

7. I am aware that there was a lawsuit predating the current lawsuit that dealt with the Dry Run Landfill in West Virginia. I know that lawsuit as the Tennant matter. During the latter part of that lawsuit, an issue arose concerning C-8. I do not recall any involvement in the Tennant matter until that issue arose. I do not recall specifically when that was.

8. I am a scientist whose day-to-day work focuses on toxicological data and reports. In Haskell Laboratory, where I work, there is an Information Section which is designated as the primary retainer of toxicological data and reports for the Laboratory. I follow a standard set of procedures for retaining documents. If I receive toxicological data or a toxicology report, which are the substantive information involved in my work, I either retain it, in hard copy form, in working files in my office or forward it to the

Information Section to be retained. I frequently do both with that type of substantive information. If I receive this type of information electronically, I print it out in hard copy and follow the above procedures. I do not recall every deleting or discarding this kind of substantive information unless I had printed it out or knew someone else was retaining it. I have a practice of providing evaluative comments about substantive toxicology data or issues verbally, and do not recall receiving someone's evaluative comments of that nature electronically. I also have a practice of cleaning my hard copy files on a yearly basis and generally use a three-year period for keeping documents for which Company policy does not require a longer retention period. If the document falls outside the three-year period, I only continue to retain it if I can find a good reason to do so from the point of view of my toxicology work.

9. With regard to documents dealing with day-to-day operations, such as correspondence, my practice is to dictate or hand-draft correspondence and then give it to my secretary to type and retain. My secretary is charged with retaining my "chron file" of correspondence and other operational categories of documents.

10. With regard to electronic mail messages that do not contain the type of substantive information described above, my standard practice is not to retain those messages. I am not very computer conversant. I prefer to communicate face-to-face, by telephone or by receipt of hard copies of documents. I prefer to receive hard copies of documents even if that means I have to wait three days to see them. Those who work with me know this and attempt not to communicate with me by email. Nonetheless, I receive a fair number of emails a day, most of which are non-substantive

communications about setting up meetings, etc. or messages sent to numerous individuals on which I happen to be included in those copied on the email. I find email intimidating, frustrating and overwhelming. It never goes away. I know how to reply to, forward, send or delete emails, but I have never electronically filed any emails. As a standard practice, I attempt to delete email that does not contain substantive information needed for my toxicology work promptly so that I do not have to sort through a backlog of email every time I look at my computer. For example, when I receive an email about scheduling of meetings, I respond electronically and then delete the received message. If I am to attend the meeting, I write the date on a hard copy calendar.

11. I have a general recollection that about a year ago I had one or more conversations with John Bowman, an in-house counsel for DuPont involved in the Tennant matter and the current lawsuit. I do not recall specifically when the conversation(s) occurred or exactly what was said. I did, however, understand from that conversation(s) that the DuPont legal department, which was handling the production of documents in the lawsuit(s), would be capturing my emails so that I did not have to retain them myself. I continued to have that understanding until June 2002 when a question was raised about retention of emails. I then learned for the first time that those involved in the production of documents were not able to capture my emails on an ongoing basis the way I understood they would. Prior to that, nothing ever arose to lead me to question the understanding I had from the conversation(s) with John Bowman. Recently, procedures have been put in place to have my secretary ensure

that emails are retained. On a going forward basis, I have suspended my standard practice of deleting emails that do not contain substantive toxicology information.

FURTHER AFFIANT SAYETH NOT.

Gerald Kennedy

COUNTY OF NEW CASTLE,
STATE OF DELAWARE, TO WIT:
On this 5th day of July, 2002, before me personally came Gerald Kennedy, to me known and known to me to be the same person described in and who executed the foregoing instrument, and he duly acknowledged to me that the matters and facts set forth herein are true and correct to the best of his information, knowledge and belief.
As witness, my hand and Notary Seal.

Notary Public

My Commission expires: 09-14-03

NOTARIAL SEAL
JAMES J. PIROLLO
STATE OF DELAWARE
MY COMMISSION EXPIRES SEPT. 14, 2003

Facsimile Cover Sheet

To: [redacted]
Company: DuPont Haskell Laboratory
Phone: 302-366-5259
Fax: 302-366-5207

From: Roger G. Perkins, Ph.D., DABT
Company: 3M Toxicology Services Medical Department
Phone: 612-733-3222
Fax: 612-733-1773

Date: 1/12/95
Pages including this cover page: 17

Gerry, Attached are the 3M references (~105) on FC-143 and your list from June '94. I started to go through the two lists and see what was missing from yours that was on our list, but concluded this is a clerical function that perhaps your information folks could do more economically than I can.

I am also suggesting a modified introduction to the data to include the following:

Ammonium Perfluorooctanoate (APFO) and "C-8" are two of the more common short-hand terms used to refer to FC-143 FLUORAD Brand Fluorochemical Surfactant, however the typical composition of FC-143 per the current 3M MSDS is as follows:

AMMONIUM PERFLUOROOCTANOATE	3825-26-1	93.0 - 97.0
AMMONIUM PERFLUOROHEPTANOATE	6130-43-4	1.0 - 3.0
AMMONIUM PERFLUOROPENTANOATE	68259-11-0	1.0 - 3.0
AMMONIUM PERFLUOROHEXANOATE	21615-47-4	0.1 - 1.0

The mixture contains straight chain perfluorocarboxylic acids, which are the desired products of synthesis as the CAS numbers listed above indicate. The straight chain homologs are the predominant specifics. There are also branched chain materials in FC-143 that might best be described by the general terms of AMMONIUM PERFLUOROISOOCTANOATE, AMMONIUM PERFLUOROISOHEPTANOATE, AMMONIUM PERFLUOROISOPENTANOATE and AMMONIUM PERFLUOROISOHEXANOATE..

It is important to keep this composition information in mind; because the published literature incorrectly refers to ammonium perfluorooctanoate and the CAS number for the straight chain 8-carbon homolog when in fact the product FC-143 is the "test substance" used for most of these toxicity studies. To date we have not documented that the straight chain eight carbon substance has been isolated and used in any toxicology studies.

GK000772

Diane R Shomper
11/05/2001 10:37 AM

To: Barry L Hudson/CL/DuPont@DuPont
cc: Michael W Bowley/AE/DuPont@DuPont, R Clifton Webb/AE/DuPont@DuPont, David M Rurak/AE/DuPont@DuPont
Subject: DRAFT Correction/Clarification to Fayetteville Observer Article

Barry --- I've tried to reach you this morning to discuss the need for a clarification in the Fayetteville Observer of your statement that Fayetteville uses a process that does not require APFO. The problem with the statement is that, on the surface, it is inconsistent with our (and the industry) position with EPA and other regulators that there are no currently no viable alternatives to APFO. Since EPA and WVDEP are aware of our work at Fayetteville, we want to make sure we go on record in clarifying your statement opposite our position with these agencies.

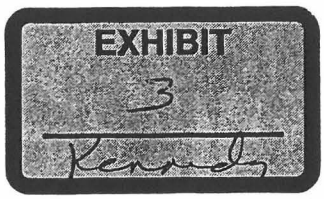
I would appreciate you and Mike Bowley taking a look at this draft and getting comments back to me today. The letter would come from you. Thanks -- Diane

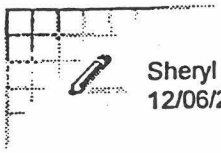
Dear Editor:

I would like to clarify a statement that appeared in the November 2 Fayetteville Observer's coverage of DuPont's announcement that it was investing $23 million to manufacture amonium perfluorooctanoate, an essential process aid in the manufacture of fluoropolymers, at the Fayetteville plant.

In the article I am indirectly quoted as saying that the Fayetteville plant uses a fluoropolymer manufacturing process that does not use ammonium perfluorooctanoate. While that is true, it should be clarified that the process used at Fayetteville is still a developmental, non-commercial process that is not viable for the broad range of fluoropolymers produced today by DuPont and others. While we expect to manufacture fluoropolymers for commercial use at Fayetteville in the near future, production will be limited to a few very select materials until the technology is further developed and proven out.

Meanwhile, the fluoropolymer process using ammonium perfluorooctanoate remains the only broadly viable process available to industry today.





Sheryl L Bradford
12/06/2000 02:16 PM

To: Paul R Kaiser/AE/DuPont@DuPont, Ronald K Amadio/AE/DuPont@DuPont, Joseph A Winkelspecht/AE/DuPont@DuPont, Glenn W Simpson/AE/DuPont@DuPont
cc: Michael J Daly/AE/DuPont@DuPont, Timothy N Clawson/AE/DuPont@DuPont, John J Plum/AE/DuPont@DuPont, Robert C Sheldon_Jr/AE/DuPont@DuPont
Subject: 1205 bldg C-8

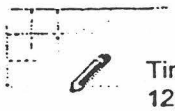
We anticipate putting "C8" (which has undergone a name change to "DFS-1") into the system on Tuesday 12/12. Only the southern half of A-bay (both floors) will be restricted to people who have been cleared to work on C8. The sump outside of A-bay (south end) will also be restricted). The restrictions are focused on loading, unloading and discharging to the sump. The restricted areas will be roped off to avoid confusion. Air monitoring was performed last year, and these restrictions are based on those results (last year we isolated the entire bay, so there will be questions as to why we aren't fully isolating again. Monitoring results showed that we don't need to be that restrictive). We will continue to perform air monitoring and modify the restrictions based on the additional results if needed.

John - please respond if I've left anything out.

Any questions, let me know.

Sheryl

---------- Forwarded by Sheryl L Bradford/AE/DuPont on 12/06/2000 02:06 PM ----------



Timothy N Clawson
12/06/2000 12:20 PM

To: Sheryl L Bradford/AE/DuPont@DuPont, John J Plum/AE/DuPont@DuPont
cc:
Subject: 1205 bldg C-8

I have responded back to Bob and reviewed our plan for non DFS-1 folks that access to the bay will not be restricted except at certain times during start up and packout/loading that will be well defined as to timing. His concern is around shift Electricans but Fire Chiefs may need more of an update as they cover off shifts for all groups.

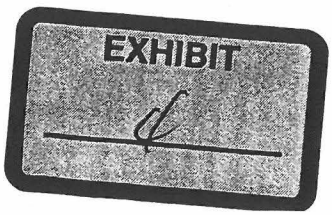
---------- Forwarded by Timothy N Clawson/AE/DuPont on 12/06/2000 07:43 AM ----------

Robert L Tirpack 12/05/2000 04:36 PM

To: Timothy N Clawson/AE/DuPont@DuPont

JJP000478

EID110386



cc: William J Ditzler/AE/DuPont@DuPont, Howard J Johnson/AE/DuPont@DuPont, Leonard J Sweeney/AE/DuPont@DuPont, Frank A Tripet/AE/DuPont@DuPont, Paul R Kaiser/AE/DuPont@DuPont, Ronald K Amadio/AE/DuPont@DuPont, Joseph A Winkelspecht/AE/DuPont@DuPont, Glenn W Simpson/AE/DuPont@DuPont

Subject: 1205 bldg C-8

Please let me know when you will be running C-8. in "A " BAY

The shift guys should not being going there without being checked at medical first.

thanks

Confidential Exhibit Removed - -
Subject to Protective Order

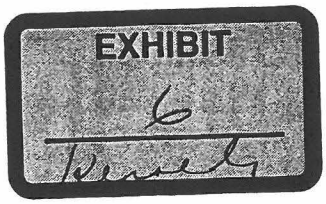
APFO
Exposure Standards

DuPont **AEL = 0.01 mg/m^3** (8-hour TWA), Skin

DuPont Community Exposure Guideline (air)
(CEG_a) = 0.3 μg/m^3

DuPont Community Exposure Guideline (water)
(CEG_w) = 1μg/L

ACGIH TLV® = 0.01 mg/m^3, skin



APFO CEG Workplace Exposure Limits

- At CEG, person exchanges 20 m^3 air/day
  20 m^3 x 0.0003 mg/m^3 = 0.6 mg or 6 µg/day

80% from air	20% from water
4.8 µg	1.2 µg
0.00024 mg/m3	Drink 2L/day
	1.2 µg/2L = 0.6 µg = 1 ppb

- If total contribution from water
  6 µg/day/2L = 3 µg/L = 3 ppb

September 2001

GK(p)001386

APFO
Workplace Exposure Limits
(AEL; TLV)

- Acute Toxicity - Moderate/Low
- Repeated Exposure Toxicity - Liver Target Organ/Effects Reversible
  (Rat - Inhalation NOEL 1 mg/m^3)
- Genetic Toxicity - Not Active
- Developmental Toxicity - Fetus not more sensitive than mother
- Reproductive Toxicity - Functional appears normal,
  structural not affected
- Carcinogenicity - Liver/testes - Human relevance unlikely
  Pancreas - potency/research to clarify

September 2001

APFO
Workplace Exposure Limits
(TLV; AEL)

Workplace limit from inhalation NOEL of 1 mg/m^3

TLV + DuPont AEL = 0.01 mg/m^3

September 2001

APFO Community Exposure Guidelines

Community Guide - 0.003 mg/m^3

Workplace Limit (0.01 mg/m^3) revised for

1) Time of exposure 168/40 hr
2) Population at risk
   - A) Aged
   - B) Very young
   - C) Childbearing age
   - D) Too ill to work

September 2001

C-8

Guidance Levels

CEG 0.0003 mg/m^3

From AEL of 0.01 mg/m^3

Factor in: 24 hr vs 8 hr

sensitive subpopulation vs worker

Reduce # by 30 x

CEGw 1 μ/L

From CEG of .0003 mg/m^3

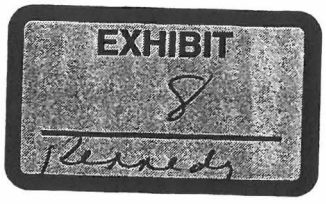
x 20 m^3/day = 0.006 mg/day

6 μg x 80% air

20% water = 1.2 μg

In 2L water/day = 1.2 μ/2L = 0.6 ≈ 1 μg/L

Key Piece: Daily acceptable intake = 6 μg/day



GK(p)001276

C-8

Guidance Levels

AEL 0.01 mg/m^3 = 0.6 ppb

Date Pieces: Oral LD_{50} rat 470 mg/kg
Inh 4 hr ALC rat 800 mg/m^3
Irritation: Moderate eye, skin
Target organ inhalation 84 mg/m^3 - lethality, liver
11 mg/m^3 - liver
7 mg/m^3 - liver
1 mg/m^3 - NOAEL
Cancer: Liver, testis, pancreas 300 ppm rats (30 LOAEL)
Genetic: Ames, saccharomyces - negative
Developmental: Rat oral 0.03/1.5/54/150 mg/kg
Rabbit oral 1/5/50 mg/kg
Rat inh 0.14/1.2/10/20 mg/m^3
No terata
Maternal = fetal
Biochemistry: Biopersists rat/mouse/…man

Key Data: 1 mg/m^3 inhalation NOAEL
30 ppm LOAEL 2 yr feeding (10 mg/m^3 inh. equivalent)
Slow clearance from blood

C-8

Guidance Levels

CEG 0.0003 mg/m3
From AEL of 0.01 mg/m3
Factor in: 24 hr vs 8 hr
sensitive subpopulation vs worker
Reduce # by 30 x

CEGw 1 μ/L
From CEG of .0003 mg/m3
x 20 m3/day = 0.006 mg/day
6 μg x 80% air
20% water = 1.2 μg
In 2L water/day = 1.2 μ/2L = 0.6 ≈ 1 μg/L

Key Piece: Daily acceptable intake = 6 μg/day

Watze de_Wolf

06/13/2002 08:04:44 AM

To: David M Rurak/AE/DuPont@DuPont, Robert C Buck/DuPont@DuPont, Stephen H Korzeniowski/DuPont@DuPont, Nils Hofman/EUR/DuPont@DuPont, Eric A van_Wely/EUR/DuPont@DuPont
cc: Matthew C Koenings/EUR/DuPont@DuPont, Gerald L Kennedy/AE/DuPont@DuPont, Harm Benjamins/EUR/DuPont@DuPont
Subject: first draft Perfluorinated surfactants

Folks,

please find attached the draft UvA report for RIKZ. It has not been reviewed by the latter organisation yet, and I unfortunately did not have time to pre-process this large report for you either (eg you'll have to do most of the 108 pages reading yourselves). The deadline for the final report from UvA to RIKZ is June 28. Floris cannot guarantee that he will incorporate all suggestions received from us.

Time for comments from DuPont to UvA is June 22, so more time then we expected earlier. Can I suggest that you submit your comments to me **June 20** the latest (COB Wilmington)? This will allow me to consolidate them on Friday June 21, and then send them in the evening to Floris.

Kind regards,
Watze

---------- Forwarded by Watze de_Wolf/EUR/DuPont on 2002/06/13 09:50 AM ----------

"F.M.Hekster" <fhekster@science.uva.nl> on 2002/06/12 03:51:46 PM

To: Watze de_Wolf/EUR/DuPont@DuPont
cc:
Subject: first draft Perfluorinated surfactants

Beste Watze,
bijgevoegd vind je de eerste versie van het stofdocument PFS. Het RIKZ heeft dit nog niet beoordeeld. Graag maak ik gebruik van jullie aanbod om dit document te reviewen. Zoals afgesproken kan ik echter geen garanties geven voor het verwerken van jullie commentaar.
Aangezien de definitieve versie van dit document de 28ste juni aan het RIKZ aangeboden moet worden, hoop ik dat jullie voor 22 juni hiernaar gekeken kunnen hebben. Mijn excuses voor deze veel te korte termijn. Ik hoop dat het desondanks haalbaar is.

Met hartelijke dank,

Floris

F.M. Hekster, M.Sc.
Environmental & Toxicological Chemistry (MTC)
Institute for Biodiversity and Ecosystem Dynamics (IBED)
University of Amsterdam
Nieuwe Achtergracht 166

EXHIBIT 9

GK(p)002413

NL-1018 WV Amsterdam
The Netherlands
+31205256578

Perfluorinated surfactant first c

Ministerie van Verkeer en Waterstaat

Directoraat-Generaal Rijkswaterstaat

Rijksinstituut voor Kust en Zee/RIKZ

FIRST DRAFT- CONFIDENTIAL

Perfluorinated surfactants

Environmental assessment

Report RIKZ/2002.xxx

1 July 2002

Authors:

University of Amsterdam
F.M. Hekster
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R.W.P.M. Laane

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Fax 31 70 3114330

Contents

Preface

In the framework of the project 'Investigating for chemicals in the future', the North Sea Directorate has put the department of Rijkswaterstaat, Institute for Coastal and Marine Management (RIKZ) in charge, to start a study on unknown chemicals. The object of this project is to identify the most important contaminants, which present a threat to the North Sea and the identification of gaps in policy, management and knowledge. In the project monitoring data are evaluated and a number of 'new' substances are proposed as a potential threat for the North Sea.
In January 2002, the University of Amsterdam, Institute for Biodiversity and Ecosystem Dynamics (IBED), Environmental and Toxicological Chemistry (MTC) has received the order to make a study on perfluorinated surfactants. This study will be directed on the whole track of perfluorinated surfactants in the environment. From production and emission to immission, waste and effects.
The project is coordinated by A.M.C.M. Pijnenburg and R.W.P.M. Laane of RIKZ. The authors of the report are: F.M. Hekster and W.P. de Voogt.

The authors wish to thank all collaborating researchers, industrial representatives and users of perfluorinated surfactants for their data sharing and highly appreciated co-operation.

Summary

General

Perfluorinated surfactants comprise a group of chemicals, containing a perfluorinated alkyl chain and an active, hydrophilic group. There are two major production routes for PFS: Electrochemical fluorination and telomerisation. The products from the first process contain a sulfonyl group (the so-called *ECF-products*); the products from the second production process contain an ethylene group (*telomers*). POSF ($C_8F_{17}SO_2F$) is the most important production intermediate for electrochemical fluorination. 8:2 FTOH ($C_8F_{17}C_2H_4OH$) is the central substance for telomer production.

Both ECF-products and telomers have four major forms of appearance, namely monomeric, homo-polymeric, co-polymeric, and phosphate esters. Co-polymers, based on acrylates or methacrylates, are the most used form of appearance. All major chemical suppliers produce or sell PFS. Until the 3M company decided to phase out their PFS production line, they were key players. Other important suppliers are Dupont, Asahiglass, Clariant, Daikin and Ciba.

For this study 15 perfluorinated surfactants have been selected. These substances are used in commercial products, monomers for polymers, important production intermediates or important degradation products. PFS have special physical and chemical properties, including chemical inertness, high thermal stability, low surface energy, hydrophobicity and oleophobicity. These properties make PFS valuable compounds for a wide variety of applications, including carpet, textile, leather and paper and board protection, fire-fighting foams, specialty surfactants.

Sources and emissions

The use and associated emissions from these applications are assessed. Paper and board is the most important applications in the Netherlands, but all this paper is imported. The carpet, leather and presumably the textile industry are the biggest users of PFS based products in the Netherlands (see table S.1).

Several applications can lead to emissions of PFS. The most important emission is the emission due to wear of PFS treated tissue (carpet, textile, leather). These emissions are polymeric; whether this can lead to monomeric PFS is not known. The use of fire-fighting foams for calamities or training leads to emissions of monomeric PFS to the environment. Furthermore, emissions from fluorochemical production sites may be a route of introduction of PFS to the environment.

Type of industry	Use of PFS (tonnes/year)	Form	Emissions (tonnes/year)
Carpet	15	Polymers	10 (worst case)
Paper & Board	*60-105* (not in NL)	Phosphates	
Textile	N.A.	Polymers	100% of the applied polymers
Leather	10-20	Polymers	
Fire-fighting foams (mobile)	0.13-0.81	Monomers	0.13-0.81 (worst case)
Fire-fighting foams (stationary)	1.0-3.0	Monomers	1.0-3.0 (worst case)
Specialty surfactants	N.A.	Monomers	-
Polymerisation aid	< 1	Monomers	-

Table S.1 Use and emissions of PFS in the Netherlands. N.A. = not available.

Behaviour in the aquatic environment
For the assessment of the behaviour of PFS in the environment many data are lacking. The available data show that the standard concepts of environmental modelling are not applicable. PFS distribution is not based on hydrophobic and hydrophilic interactions, but on among others, electrostatic interactions. PFS does not accumulate in fat, but binds to the macromolecules in blood plasma and liver.

n-EtFOSE, n-MeFOSE and n-EtFOSA (ECF-products) and 6:2 FTOH, 8:2 FTOH and 10:2 FTOH can escape from the water phase to air, considering their relatively high Henry constants. This tendency is supported by their detection in Canadian air. This can be an important factor in the global distribution of PFS. Other fluorinated chemicals have lower Henry constant and are expected to stay in the water phase.
PFOS and 8:2 FTOH exhibit a high sorption potential and desorption is difficult.

Test results show that the perfluoroalkyl chain of ECF-products is not affected by biodegradation, hydrolysis or photolysis. The non-fluorinated part is expected to be degraded to form PFOS or PFOA. The degradation products of telomers are not known, but it is expected that the perfluorinated chain is not affected by degradation, hydrolysis or photolysis. 8:2 FTOH was shown to be transformed in rats to PFOA. For fluorinated polymers no degradation data are available.

PFOS is highly bioaccumulative, considering its bioaccumulation factor of 6300-125000. PFOA hardly bioconcentrates (BCF = 1.8) and 8:2 FTOH has a bioconcentration factor of 87-1100.

Occurrence
PFOS and to a lesser extent PFOA were detected in the environment on a global scale. Point sources can lead to elevated levels of PFS in biota and the abiotic environment. Concentrations of PFS are higher in more urbanised or industrialised areas, in biota and in the abiotic environment.
Concentrations in biota from North America were highest, followed by biota from Europe. Concentrations in biota from remote locations as the Arctic were much lower. All PFS that were detected in biota were present in blood, liver, kidney, muscle or brain. No data are available for the occurrence of telomers in the environment.
In humans, PFOS and PFOA was detected in occupationally exposed workers and in the general public. Levels in fluorochemical production workers were 0.135-2.44 ppm (PFOS) and 0.106-6.8 ppm (PFOA); concentrations in the general public were 17-53 ppb (PFOS) and 3-17 ppb (PFOA).

Toxicity
Many toxicity tests for PFOS and PFOA have been performed with limited reliability. The reliable results show that PFOS is moderately acute toxic to freshwater fish and invertebrates. Toxicity to algae is relatively low. The chronic toxicity of PFOS to freshwater fish and invertebrates is moderate. PFOS is moderately toxic to marine invertebrates (acute and chronic) and algae (acute).
The derived $iMPC_{freshwater}$ is 6 •g/L. PFOS concentrations were shown to exceed the iMPC, in point source receiving fresh water. In other freshwaters, the iMPC was approached.
The acute toxicity of PFOA to freshwater invertebrates and algae is moderate, whereas the toxicity to freshwater fish is relatively low. An $iMPC_{freshwater}$ for PFOA of 3.8 •g/L has been derived. This iMPC can be exceeded due to point sources.

For telomers no conclusions regarding their toxicity can be drawn.

Concerning humans, both PFOS and PFOA have long half-lives (8.67 and 1-3.5 years, respectively) in the human body. Both chemicals are distributed to liver, plasma and kidney. To rodents PFOS and PFOA exhibit low acute toxicity, but they are eye irritating.

In chronic feeding tests with rodents and primates the primary target was the liver for PFOS and PFOA. PFOA was found to be weakly carcinogenic. Mutagenicity testing of PFOS did not show any mutagenic effects. PFOA did not show mutagenic effects in most mutagenicity test, but did induce chromosomal aberrations and polyploidy in CHO cells.

In a developmental effect study with PFOS the NOAEL and the LOAEL for the second generation of rodents were determined to be 0.1 mg/kg/day and 0.4 mg/kg/day, respectively.

Policy

In the Netherlands, no specific policy concerning PFS exists. In the USA the production and import of some ECF-products is regulated and a hazard assessment on PFOA was performed. The governments from Canada, the United Kingdom and Denmark show awareness for the risks of PFS. Furthermore, the OECD executed a hazard assessment on PFOS.

The 3M corporation has performed various studies on the toxicology, pharmaco-kinetics and environmental fate and effects of ECF-products. The manufacturers of telomers, gathered in the Telomer Research Program (TRP), have set up a research program on the toxicology, pharmaco-kinetics and environmental fate and effects of 8:2 FTOH.

1 Introduction

1.1 Background

In 1993 and 1996 papers and reports were published on the exposure of humans to perfluorooctanoic acid (PFOA) (Gilliland & Mandel, 1993; Gilliland & Mandel, 1996). A few years later several publications in the environmental literature have given attention to perfluorinated surfactants (PFS) (Key et al, 1997; Moody & Field, 1999). This attention was made possible by improved analytical techniques, resulting in the characterisation of this group of chemicals in environmental samples. Perfluorooctane sulfonate (PFOS) has been detected all around the globe, both in animals and in humans (Olsen et al, 1999; Giesy & Kannan, 2001). These data did have consequences for the chemical industry. On May 16, 2000, 3M announced that it was phasing out the perfluorooctanyl chemistry production; the decision was based on '*[..] principles of responsible environmental management.*' (3M, 2000).

Although no adverse effects had been observed at the detected concentrations, this decision and its reasons resulted in international attention and awareness in scientific and non-scientific media (AtoFina, 2000; Browne, 2000; USEPA, 2000a; Wood, 2000; Clarke, 2001, Renner, 2001).
Furthermore international research projects have been started on the environmental behaviour of perfluorinated chemicals. In June 2002, draft hazard assessments are available for PFOS (OECD, 2002) and PFOA (USEPA, 2002). Furthermore, a large international research program is executing studies on the environmental and toxicological properties of 1H,1H,2H,2H-Perfluorodecanol (8:2 FTOH, TRP, 2002).

1.2 Objectives

The objectives of this study with regard to perfluorinated surfactants are:
To give an analysis of the problems in the aquatic environment: a description of the load, occurrence, behaviour and effects and a analysis of the problems which indicate how the presence of perfluorinated surfactants may disturb the functioning of the different water systems by effects on sensitive organisms. Furthermore giving an overview of the national and international policy.

In this study the most recent information on perfluorinated surfactants has been used. PFS are under much international scientific attention. This results in continuous publications on this matter. This document tries to reflect the state of knowledge in June 2002.

The study has a broad set-up. The next aspects will be handled. In chapter 2 the chemical characteristics perfluorinated surfactants are described. In chapter 3 the production process is clarified and the use and associated emissions of these chemicals to the environment are described. In chapter 4 the behaviour in the environment is described, followed by chapter 5, dealing with the occurrence in the environment. In chapter 6 and 7 an overview is given of the toxicity data and the policy, respectively.

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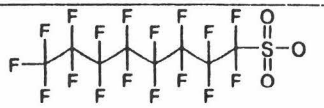
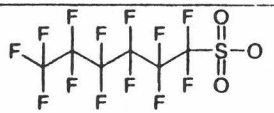
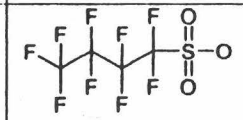
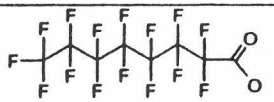
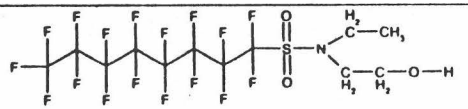
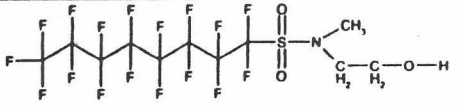
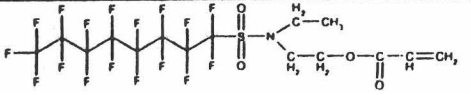
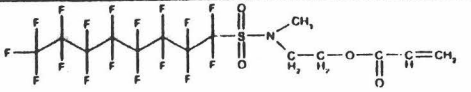
For many of these substances very few physical-chemical data are available. This study will focus on the most important commercial products, the primary production intermediates, and the major degradation products.

PFS can be produced via two distinct routes of synthesis:
- Simons Cell Electrochemical Fluorination (ECF), as used by 3M and Miteni,
- Telomerisation, as used by among others Dupont and Atofina

The production processes will be discussed in the next chapter. The ECF process yields perfluorinated products with a sulfonyl group. The products from telomerisation are not perfluorinated, but have a perfluoroalkyl chain with an ethylene group followed by a functionalised group (see paragraph 3.2).

The products that are produces via the two routes of synthesis have four major forms of appearance, being monomeric, homo-polymeric, co-polymeric and phosphates esters. The form of appearance is dependent on the application, with co-polymers as the most used one. The various applications and chemicals involved are described in more detail in chapter 3.

Fluorinated polymers are mostly co-polymers of fluorinated acrylates. They can be considered to be fluorosurfactants. However, polymers exhibit a totally different environmental behaviour than low molecular weight compounds. Furthermore, very few properties are known of polymeric fluorosurfactants, including fluorophosphates. Therefore the polymers will be treated in a different way in this study. They will not be included in the table of primary study substances, but their production intermediates will be. The degradation products or production impurities from fluorinated polymers can be low molecular weight fluorosurfactants. These will be incorporated in this study.
In products the fluoroalkyl chain length can vary from four up to twenty. In general, most products have chain lengths of between six and ten. Most data are available on the chemicals with eight carbons. Therefore, this study will focus on the following products (see table 2.2):

Abbrevation	Full name	Sel. Criterium*	CAS-number	Structure
PFOS	Perfluorooctyl sulfonate	1	Various salts	
PFHxS	Perfluorohexyl sulfonate	1	Various salts	
PFBS	Perfluorobutyl sulfonate	1	29420-49-3	
PFOA	Perfluorooctanoic acid	1,3	Various salts	
n-EtFOSE	n-Ethylperfluorooctanesulfonamidoethanol	2	1691-99-2	
n-MeFOSE	n-Methylperfluorooctanesulfonamidoethanol	2	24448-09-7	
n-EtFOSEA	n-Ethylperfluorooctanesulfonamidoethyl acrylate	4	423-82-5	
n-MeFOSEA	n-Methylperfluorooctanesulfonamidoethyl acrylate	3, 4	25268-77-3	

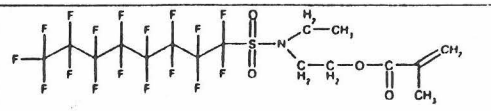
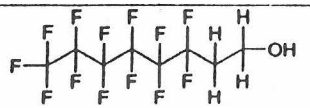
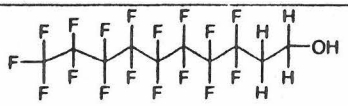
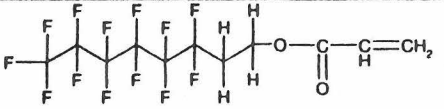
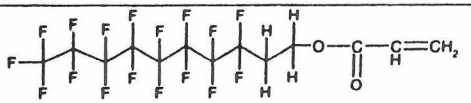
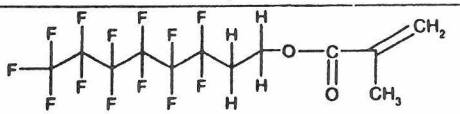
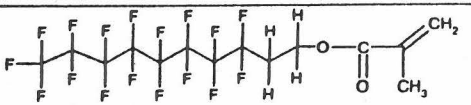
n-EtFOSEMA	n-Ethylperfluorooctane sulfonamidoethyl methacrylate	3, 4	376-14-7	
6:2 FTOH	1H,1H,2H,2H-perfluorooctanol	2	647-42-7	
8:2 FTOH	1H,1H,2H,2H-perfluorodecanol	2	865-86-1	
6:2 FTA	1H,1H,2H,2H-perfluorooctyl acrylate	4	17527-29-6	
8:2 FTA	1H,1H,2H,2H-perfluorodecyl acrylate	4	27905-45-9	
6:2 FTMA	1H,1H,2H,2H-perfluorooctyl methacrylate	4	2144-53-8	
8:2 FTMA	1H,1H,2H,2H-perfluorodecyl methacrylate	4	1996-88-9	

Table 2.2. Primary study substances. * The selection criteria are (1) Important degradation product (2) Important production intermediate (3) Important commercial product (4) Important monomer for polymers

The above-mentioned substances have various names. In general they are referred to as fluorosurfactants, perfluorosurfactants, perfluorinated surfactants, fluorinated surfactants, fluorinated chemicals, perfluorinated chemicals, or fluorochemicals. Some people speak about 'POSF-based' or 'POSF-related' substances, in contrast to fluorinated telomer-products. The products that are synthesis via telomerisation are also referred to as telomers. The commercial names for perfluorinated chemicals can be found in table 3.7.

Fluorinated polymers or polymeric fluorosurfactants are not to be confused with fluoropolymers.

2.2 Physical-chemical characterisation

Some of the substances in Table 2.2 are process intermediates, others are used themselves in formulations and some of these products only occur due to degradation processes. For the majority of these substances no physical-chemical properties are available. For the polymers no data are available at all. Furthermore, the reliability of some of the available data is doubtful. To describe the reliability of the data a Data Reliability Indicator (DRI) is used, as developed by Klimisch et al (1997). In Annex 2 the methodology of the DRI is explained.
In table 2.3 the available, reliable data are collected:

Substance	Molecular weight (g/mol)	Melting point (°C)	DRI[a)]	Boiling point (°C)	DRI	Water solubility	DRI	Vapour pressure (Pa)	DRI	H (atm*m^3*mol^{-1})
PFOS (K^+)	538.23	> 400[1]	1	-	-	519 mg/L[2] 570 mg/L[3]	1 N.D.	3.31 E-4[4]	1	3.4 E-9 (calc.)
PFHxS	438.22	-	-	-	-	-	-	-	-	-
PFBS	338.21	-	-	-	-	51 g/L[5]		-	-	-
PFOA (NH_4^+)	431.1	Sublimes at 130°C[6]	-	Sublimes at 130°C[6]	-	> 500 g/L[5] > 100 g/L[6]	- -	<1.3 mPa[5] 9.2 mPa[6]		< 1.1 E-11 (calc) 7.8 E-11 (calc)
n-EtFOSE	571.26	55-60[7]	2	-	-	151 µg/L[8]	1	0.504[9]	2	1.9 E-2 (calc)
n-MeFOSE	557.23	-	-	-	-	-	-	-	-	-
n-EtFOSEA	625.30	27-42[10]	2	150 at 1mm[10]	2	0.89 mg/L[10]	2	-	-	-
n-MeFOSEA	611.28	-	-	-	-	-	-	-	-	-
n-EtFOSEMA	639.33	48-55[11]	-	-	-	-	-	-	-	-
6:2 FTOH	364.11	-	-	88-95 at 28mm[12]	-	12-17 mg/L[13]	-			~ 1 E-2[13]
8:2 FTOH	464.12	49-51[14]	-	112-114 at 10mm[14]	-	140 µg/L[13]	-	2.93[15]	-	9.6 E-2[13]
6:2 FTA	418.16	-	-	-	-	-	-	-	-	-
8:2 FTA	518.17	-	-	90 at 4mm[16]	-	-	-	-	-	-
6:2 FTMA	432.18	-	-	-	-	-	-	-	-	-
8:2 FTMA	532.20	-	-	120 at 4mm[17]	-	-	-	-	-	-

Table 2.3. Properties of selected fluorochemicals. a) DRI = Data Reliability Indicator (1) 3M, 1999a (2) 3M, 2000 (3) 3M Reports, 1999 (4) 3M, 1999b (5) Miteni, 2002 (6) APME, 2002 (7) 3M, 1999c (8) 3M, 2001 (9) 3M, 1998, (10) 3M, 1996 (11) Fischer Scientific, 2001(12) ABCR, a (13) Dupont, 2002 (14) ABCR, b (15) TRP, 2002 (16) ABCR, c (17) ABCR, d. Calc = calculated. N.D. = not determined.

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3 Production, Use & Emissions of PFS in the Netherlands

3.1 Introduction

PFS are used in numerous applications. Because fluorinated surfactants are relatively expensive, they are only used when other products do not possess the specific requirements (Kissa, 2001). Perfluorinated surfactants have special physical and chemical properties, including chemical inertness, high thermal stability, low surface energy, hydrophobicity and oleophobicity (Smart, 1994, Kannan et al, 2001). These characteristics make them valuable compounds in several fields of application.
The most important fields of application are (USEPA, 2002, NCEHS, 2001, Dupont, 2002):

- Carpet protection,
- Paper and board protection,
- Textile protection,
- Leather protection,
- Fire-fighting foams,
- Specialty surfactants,
- Polymerisation aid.

The distribution over use categories in the Netherlands is not precisely known. A recent inventory of the use of PFS in the United Kingdom, shown in table 3.1, shows the relative importance of the several categories. Table 3.1 also presents the breakdown in global application categories of the perfluorinated products of the 3M company.

Application area	Use UK (NCEHS, 2001) (%)	Application area	Global 3M production (OECD, 2001) (%)
Carpet & Textile Treatment	48.8	Surface treatment	48
Paper & Board Treatment	15.0	Paper protection	33
Speciality Surfactants	17.5	Performance chemicals	15
Fire-Fighting Chemicals	16.3	Fire-fighting foams	3
Chemical Intermediates	2.5		

Table 3.1. Proportional breakdown of perfluorochemical use in the UK and global 3M production.

From these data it is obvious that carpet and textile treatment constitute the major use category, probably followed by paper treatment. Although this breakdown can be different for the Netherlands, because of the difference in relative importance of industry branches from country to country, it is expected that at least some of these use categories will be important users of PFS in the Netherlands.

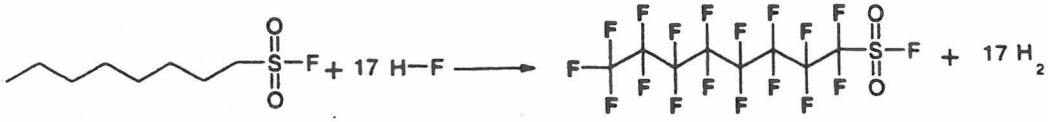
The applications and their corresponding emissions to the environment will be discussed in this chapter. Other applications such as herbicide, cosmetics, and electronics will not

be discussed, because they are used in smaller quantities. Kissa (2001) reviewed most of the possible applications for fluorochemicals.
Another possible source of fluorinated chemicals to the environment are the emissions from fluorochemical production sites.

For all applications a few routes of emissions are possible. The emissions result from production, from use, from collected and uncollected waste after use (both monomeric and polymeric), as well as from waste treatments (incineration in a municipal waste incinerator, water purification in a waste water treatment plant (WWTP)).

3.2 Production

There are two major commercial production processes for PFS: electrochemical fluorination (ECF) and telomerisation.
In the ECF process an organic compound is dissolved or dispersed in anhydrous hydrogen fluoride. A direct electric current is passed through the hydrogen fluoride, causing all the hydrogen atoms to be replaced by fluorine. The overall reaction is as shown in figure 3.2:



1-Octane sulfonyl fluoride — Perfluoro-1-Octane sulfonyl fluoride (POSF)

Figure 3.2. Example of the ECF process

In this process fragmentation of the alkyl chain can occur. Therefore, the products of this production process contain various impurities. The process, its products and its impurities are described more extensively in Annex 3.

In the telomerisation process Iodopentafluoroethane is reacted with *n* units of tetrafluoroethene (TFE); the reaction with 3 units is shown as an example in figure 3.3: This production process yields straight chains, with hardly any impurities, but the

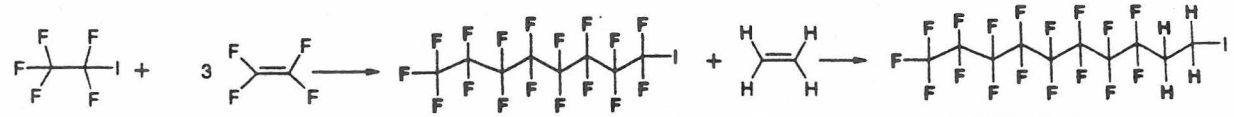


Figure 3.3. Example of the telomerisation process

products are not fully perfluorinated. The ethylene group is characteristic for this production process. The process and its products are described more extensively in Annex 3.

3.3 Use and emissions

Perfluorinated surfactants are used for various applications as will be discussed in this chapter. Many suppliers manufacture and market PFS for a variety of applications. Until 3M decided to phase out their perfluorooctyl chemistry (3M, 2000a), they were the most important global producer of PFS. Recently Dupont bought the fluorinated telomer

division of AtoFina (AtoFina, 2002). Other important PFS suppliers are Asahiglass (Japan), Daikin (Japan), Clariant (Germany) and Bayer (Germany).

The different commercial names of these products are reported in table 3.7 at the end of this chapter.

3.3.1 Carpet protection

Introduction

Fluorinated surfactants are used to form a protective, soil repellent coating on carpets. The principle of soil repulsion is based on the reduction of the surface energy of the fibre by the fluoroalkyl chains. These chains repel both water and oil. Therefore soil particles cannot enter the carpet. The mechanism is explained in figure 3.4:

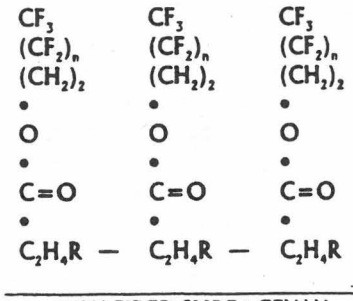


Figure 3.4. Mechanism of carpet protection with fluorinated polymers (Tomasino, 1992).

These soil repellent products are generally referred to as Scotchgard products, which is the brand name of the 3M product for this application. The commercial products for carpet protection contain approximately 15% fluoroalkyl acrylic polymers (Tomasino, 1992, 3M, 2000b, 3M, 2000c). Well-known products are Scotchgard (3M), Zonyl (Dupont), Baygard (Bayer) and Foraperle (Atofina). In general these products are used as foam-applied emulsions for the finishing of the carpets (VNTF, 2002).

Use figures

The estimated use of fluorinated polymers in the carpet industry in the Netherlands is approximately 100 tonnes of products annually. With an average of 15% fluorinated polymers (VNTF, 2002) this corresponds to 15 tonnes of fluorinated polymers. The amounts used are not at all constant; the (temporary) withdrawal of the 3M products of the market has an important influence on the fluctuations.

	1999	2000	2001
Used fluorosurfactants products (tonnes)	102.4	136	80.9

Table 3.5. Consumption of fluorosurfactants in the carpet industry in the Netherlands.

Apart from carpet manufacturing in the Netherlands, also PFS treated carpets are imported from foreign countries. For the total carpet industry 141 million m^2 is produced in the Netherlands, 125 million m^2 is exported and 75 million m^2 is imported (VNTF, 2001). Therefore in the Netherlands annually 91 million m^2 carpet is sold. For

the production of these carpets approximately 65 tonnes PFS-based products are used, with about 10 tonnes fluorosurfactants[1].

Emissions
There are several possible routes of emission of PFS from the use and consumption in the carpet industry.

1. Application in the factory
2. Wear from the carpet
3. Emission of monomers from polymers
4. Emissions from reapplication on fixed carpets
5. Emissions from the waste phase

Ad 1) In the carpet factory the fluorosurfactants-based finishing is applied and the carpet is dried afterwards. From that application emissions may occur: these cannot be quantified in this study, because emission factors are not known (GuT, 2002).
Ad 2) The durability of the protective PFS layer on carpets has been studied. ' [..] *it is expected that 50% of the FC [fluorochemical] treatment will be removed over the nine-year life of the carpet due to walking and vacuuming, while an additional 45% of the FC treatment will be removed in steam cleaning throughout the carpet life* (3M, 2000d).' These percentages might vary with different products (Dupont, 2002). The wear of polymeric fluorosurfactants with a corresponding emission of fluorinated polymers to the environment does not necessarily lead to the emission of PFS. The degradation of fluorinated polymers is not known (see paragraph 4.4.4).
In a worst case estimate all the polymer degrades to form PFS. 95% of the polymer is removed. On carpets that are used in the Netherlands, 10 tonnes of perfluorinated polymers are applied. Therefore 9.5 tonnes of PFS can be released to the environment, due to wear of carpet protection polymers.
Ad 3) The use of fluorinated polymers can lead to the emission of perfluorinated surfactants to the environment via a direct or indirect route.
The direct emission of PFS is due to impurities in the products. During the various steps of the production process, used to form functionalised products, reaction impurities are formed. The impurities represent 1-2 percent of the total production volume and will be present in the finalised product (3M, 2000e). The impurities will also be present in the monomers used for manufacturing of the polymers. It is very likely that the impurities will not polymerise. Whether the impurities will be present in the polymer as monomer is not clear. If they are, it is possible that they will be released from the polymeric product after its application to the carpet
Secondly, polymerisation is often not a fully efficient reaction. A small part of the monomer will not react and will be present in the final product as a low level residual (3M, 2000f). The monomers have a composition different from the impurities. They can also be released from the product, leading to the emission of perfluorinated functionalised products.

The indirect route of emission of PFS from polymers originates from PFS that are not present in the polymer. Physical or chemical degradation might lead to the formation and subsequent emission of PFS from the polymer. Fluorinated polymers are said to be stable (3M, 2002, Bayer, 2002), but the degradation to PFS has not been studied.

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[1] 141-125 = 16 million m^2 produced for the Dutch market. 75 million m^2 imported makes 91 million m^2 used annually. It is assumed that production processes in foreign countries used approximately the same amount of PFS for carpet protection. Therefore 91/141 * 100 tonnes makes 64.5 tonnes. 64.5 * 0.15 = 9.7 tonnes PFS.

A laboratory test with a perfluorinated polymer treated tablecloth confirmed the emission of monomers from a polymeric application. An extraction at moderate temperature (60 •C) with an organic solvent showed the possible leakage of perfluorinated monomers from the treated tissue. The origin (direct or indirect) of the PFS was not studied (Jonkers et al, 2002).
If a worst-case estimate is made, all the fluorinated polymers can be degraded or transformed to form PFS. As was calculated above, this can lead to the emission of 9.5 tonnes of perfluorinated surfactants. This is the worst-case estimate of ad 2 and 3 together.
Ad 4) Carpets that have been treated with fluorochemicals are sometimes re-treated. This can be done after cleaning by professional carpet cleaners, or by consumers, with spray applications. The consumer application does not appear to be an important application in the Netherlands. A short survey with carpet shops showed that fluorochemical protection sprays were not available (Alto, 2002, Carpetland, 2002, NTU, 2002, ITC, 2002). Carpet cleaners do use fluorochemical-based products for the application of a new protective layer (Chem-dry, 2002). This could lead to emissions. Neither the use, nor the emissions from this application could be quantified in this study.
Ad 5) Carpets that are disposed off after use will be added to the general waste stream. If the carpet cannot be recycled, it will be combusted or landfilled. In the Netherlands most of the non-recyclable waste is combusted (52%), but still an important amount of the waste is landfilled (39%)(Milieuloket, 2001). Bond-energy calculations predict that combustion will lead to the destruction of PFS (3M, 2001a, 3M, 2001b). In the leachate of landfill, PFOS and PFOA have been detected (3M, 2001c). The landfill of PFS treated products can lead to the emission of PFS to the environment.

It appeared to be complex to estimate emissions of PFS from the use and consumption in the carpet industry. The worst-case estimate for emissions of PFS from carpets that are used in the Netherlands is 9.5 tonnes. The treatment with sprays for re-application is not taken into account. These 9.5 tonnes have been applied in polymeric form and could be released by five different ways. The degradation and transformation of fluorinated polymers is the most important remaining question to improve the worst-case estimation.

3.3.2 Paper and board protection

Fluorinated chemicals are used in the paper industry to produce water and greaseproof paper. Among others, this paper is used for the wrapping of snacks, cookies and pet foods (Niermans, 2002, Pfleiderer, 2002, Proost & Brand, 2002). This type of material is generally referred to as *Ersatz* paper.
The products that are used for this application are generally based on fluoroalkyl phosphates (3M, 1999a, Kissa, 2001, NCEHS, 2001).

Proofing of paper does not take place in the Netherlands. The majority of this grade of paper present that is present in the Netherlands is imported from Germany and Scandinavia (Niermans, 2002, Proost & Brand, 2002).
The main suppliers of fluorochemicals in the paper industry are 3M, Atofina, Bayer, Ciba, Clariant and Dupont with respectively the following products: Scotchban, Foraperle, Baysize-S / Baysynthol, Lodyne, Cartafluor and Zonyl (Pfleiderer, 2002).

Use figures

No production of this grade of paper or board is known in the Netherlands (VNP, 2002). A market survey in 2000 estimated that in the Netherlands between 6000 and

7000 tonnes of *Ersatz* paper is used annually (Niermans, 2002). It is estimated that for these types of paper 1.0-1.5% (based on the dry weight of the fibre) fluoroalkyl phosphate is needed (Kissa, 2001), resulting in 60-105 tonnes of fluoroalkyl phosphate.

Emissions
Emissions of PFS due to the use of Ersatz paper are from migration out of the paper to the wrapped product (Dupont, cited in NCEHS, 2001), and emissions from paper plants in adjacent countries. The emissions from factories are believed to be very small (3M, 2000d).
Another source of emissions is the cutting waste in the paper converting industry, leading to solid waste of PFS treated paper.
In the waste phase used paper can also lead to the emission of PFS. During incineration all PFS will be destroyed, but leachate from landfills can lead to emissions to the soil and water (3M, 2001c).

3.3.3 Textile protection

Fluorinated chemicals are used extensively in the textile industry and by private consumers. The application is similar to that in the carpet industry. The products used are polymers, based on fluorinated acrylates and methacrylates, and are referred to as *fluorcarbon* (Lakatex, 2002).
The goal of the application of fluorinated chemicals is to provide water, oil, soil, and stain repulsion (NCEHS, 2001). Textiles that are used for i.e. tablecloth, upholstery, rainproof clothing and bed linen are treated with these protective chemicals. There are two stages in the production process that use fluorcarbon, both intended to form a fluorinated coating.

Use figures
The textile industry in the Netherlands comprises many small and medium enterprises. Some of these companies use fluorosurfactants in their manufacturing process. Because the industry is scattered and public data are neither available from the Vereniging Textielindustrie Nederland (*Dutch Assocation for the Textile* Industry, VTN, 2002), nor from the European Apparel and Textile Organisation (Euratex, 2002), it is not possible to estimate the use of fluorochemicals in the textile industry.
For these applications approximately 2.0-3.0% (of the fibre weight) perfluorochemicals are necessary to obtain the water repulsion (Kissa, 2001). However, the total amount of waterproof textile fabricated is not known. In the United Kingdom, textile and carpet applications together contribute for 48.8% of the fluorochemical active ingredients (NCEHS, 2001). It is likely that this industry in the Netherlands uses considerable amounts of fluorochemicals.
Textile chemicals are obtained from various manufacturers. Information from stakeholders indicates that Bayer, Dupont, 3M and Daikin are the most important suppliers and have all together a market share of approximately 90% (VTN, 2002, B.L.W. Visser, 2002). Unfortunately, sales figures are not available from the suppliers.

Emissions
There are five possible routes of emissions of PFS from the use in the textile industry and consumption:

1. Losses during application in the factory
2. Wear from the textile
3. Emissions of monomers from polymers
4. Emissions during reapplication to textiles

5. Emissions from waste treatment

Ad 1) The treatment of textiles with fluorinated chemicals in the factory leads to the emission of fluorinated polymers. There is an emission of fluorinated chemicals present in the cut-offs as solid waste. This is a very small percentage of the textile production (Lakatex, 2002). These two emissions together are estimated to form approximately 10 percent of the used fluorinated chemicals (3M, 2000d).
Ad 2) The fluorinated coating on textiles is vulnerable to wear. During the lifetime of the product a considerable part of the fluorinated polymer will be removed, analogous to the wear from carpets. For textiles the intensive washing can increase the amount of the coating that is lost to the environment. This emission has not been quantified. A worst-case approximation would estimate that 100 percent of the applied fluorochemicals are released to the environment.
Ad 3) The use of polymers can lead to the emission of monomers. Both product impurities and non-polymerised monomers can be a source of PFS to the environment. This is extensively discussed for the application to carpets in paragraph 3.3.1 and is analogously valid for the textile applications. Additionally the intensive washing of textiles can lead to the emission of monomers. In a worst-case estimation it is suggested that all the polymers degrade to form PFS.
Ad 4) On the consumer market several products are available to improve water and grease proofing of textiles. These products are available as sprays and wash-ins. Some of these products contain fluorochemicals (Bever, 2002, Denig, 2002, Grangers, 1997, Grangers, 1998). Although sales of these products are said to be considerable, no estimation of the market can be made. Both types of (re-) application of fluorinated coatings can lead directly to emissions to the environment. 3M (2001) estimated that 34% of the product that is expelled from a spray is lost to air. Evidently, the part of the wash-in application that is not properly attached to the fibre will be emitted to the sewer.
Ad 5) Textiles that are not recycled will be combusted or landfilled. Presumably, combustion will lead to the destruction of monomeric and polymeric PFS, whereas landfill can lead to the emissions of PFS to the environment (see paragraph 5.3).

3.3.4 Leather protection

Perfluorinated surfactants are used for the treatment of leather. The main function is to provide waterproofing. For this application polymeric fluorochemicals are used (Kissa, 2001).
The water repellents are used in the finishing process. Water repellent consumer sprays are also available for leather products.

Use figures

The Dutch Federation of Tanners (FNL, Federatie van Nederlandse Lederfabrikanten) is executing an inventory of the use of various products in the leather industry. Fluorinated chemicals are incorporated in this survey. Results are forthcoming (FNL, 2002).
According to Kissa (2001), concentrations of fluorochemical in the leather industry are very low (0.025-0.05% on leather weight).
Furthermore, much of the leather used in the Netherlands is imported, and much of the produced leather is exported. In 1998 the production of leather in the Netherlands was approximately 7 million m^2, export was 5 million m^2, and import was 3.6 million m^2 (FNL, 2000). The average mass of leather is approximately 6 kilograms per m^2 (UNIDO, 2000). If we assume that all the leather has been treated with 0.025-0.05% PFS (being a worst-case estimate), the total use for the Dutch leather production would be 10 – 20 tonnes of polymeric PFS.

Emissions
No emission estimates have been made for this branch of industry. Emissions are possible from the application in the factory, from fluorinated chemicals on leather waste and wear from leather during use. Land filling of leather waste can lead to the emission of PFS to the environment.
Furthermore, the spraying of leather can lead to direct emissions of PFS to the environment.

3.3.5 Fire-fighting foams

Introduction
Flammable liquid fuel fires form a serious threat to life. For the prevention of these risks, aqueous film forming foams (AFFF) were developed in the 1960s as fire-extinguishing agent for this type of fires (Moody and Field, 2000). The AFFF, when mixed with water and air, provides a fire-extinguishing film consisting of a foam.
PFS contribute to the performance of AFFF, but comprise only a relatively small fraction of the formulation (0.5-1.5%, Moody & Field, 2000, 3M, 1999b, Solberg Scandinavian, 2001). For these applications monomeric perfluorinated salts are used (Moody & Field, 2000). A detailed description of the mechanism of fire-fighting foam can be found in annex IV.

In the Netherlands no foam-forming agents are produced; these are imported (Luttmer, 1998, Ajax, 2002). The use of foam-forming fire extinguishers can be divided in two groups:

1. Mobile fire extinguishers,
2. Stationary fire-fighting systems.

The first group comprises the mobile hand-held extinguishing equipment; the second group comprises stationary fire-fighting systems, including large stocks of foam-forming concentrate.

Mobile fire extinguishers
Three types of mobile fire extinguishers exist, of which foam-forming extinguishers are becoming more and more important (Ajax, 2002):

- Powder
- Carbon dioxide
- Foam

The last few years more environmentally friendly mobile foam fire extinguishers have been introduced, and a Dutch certification scheme 'Milieukeur' has been established. There are two suppliers that have products that comply with the scheme (Milieukeur, 2002). These extinguishers contain less or no PFS and have a large market share with at least one supplier; this company sells approximately 95% so-called 'Eco-foam' (Ajax, 2002).

Use figures
It is estimated that in the Netherlands annually 150,000 mobile foam fire extinguishers are sold, with an average size of 6 litres (Ajax, 2002). For these extinguishers 54,000 litres foam forming concentrate is used annually, with 0.5-1.5% perfluorinated chemicals, being 270-810 kg PFS. Due to these use of eco-foam this use is diminished with approximately 50% (135-405 kg PFS).

Apart for new extinguishers, an important part of the foam concentrate is used for the refilling of used extinguishers and for the standardised five-yearly revision. No data are available for the estimation of quantity of this application.

Emissions

There are two important emissions due to the use of fire-fighting foam in mobile equipment: the emission during use and the emission from the disposal of old filling when refilled. Both are non-controlled (Ajax, 2002).

The emission during use, for both training and real accidents is inevitable. Dependent on the place of fire, the fire-fighting foam, with the PFS, is emitted to the environment. Foam extinguishers have to be refilled every five year. Although not all extinguisher owners do presumably follow this standard, most of the equipment is refilled. On revision the filling has to be replaced with a new filling. The old filling, with diluted foam-forming concentrate, is disposed to the sewer and treated in a sewage treatment plant. From an environmental monitoring study (3M, 2001c) it is known that PFOS is still present in the WWTP effluent. Moody & Field (2000) state that analytical methods are not accurate enough to estimate the removal efficiency for fluorinated surfactants. Furthermore, the use of AFFF can lead to problems at the WWTP. Excess foaming may occur from large discharges of AFFF. Other constituents of AFFF can lead to significant higher BODs and CODs (Moody & Field, 2000).

A worst-case approximation would estimate the release to the environment of all the AFFF purchased. This would lead to the emission of 135-810 kilograms PFS annually.

Stationary fire-fighting systems

Five types of fire-fighting agents are available for stationary systems, of which foam-containing PFS is only one. For these applications no eco-foam is available so far. Many standards have been set for these systems, including various tests; the eco-foam concentrate has not been subjected to these tests (Ajax, 2002).

In contrast with the mobile fire extinguishers, the emissions from stationary systems are much more controlled by regulation. Foam-forming concentrate, which has expired is not disposed to the sewer, but has to be collected and transported to a waste incineration plant. It was not possible to retrieve disposal data (LMA, 2002).

Use figures

At this moment no data for the total are available. In the Dutch Air Force approximately 3,200 litres AFFF concentrate are used for calamities or prevention annually. These are emitted. Until 2000 another 2,800 litres AFFF concentrate was used annually for fire-fighting training. Nowadays water is used for training (Koninklijke Luchtmacht, 2002). The fire brigade of Schiphol International Airport, Amsterdam, used AFFF for training facilities until December 2001. Nowadays they train with water. The last use of AFFF in non-military aviation in the Netherlands for calamities was in 1998. The current annual use is estimated to be close to zero. The stock of AFFF at Schiphol Airport is about 1200 litres, the filling in the equipment excluded (Schiphol Airport Fire Brigade, 2002).

A large conglomerate of companies in the Rotterdam Harbour Area '*Rijnmond*' has an annual substitution of expired AFFF of 75,000 litres concentrate. For the protection of the newly constructed railroad route '*Betuwelijn*' 150,000 litres of AFFF concentrate was purchase by the Dutch government (Ajax, 2002). The latter is an incidental purchase and is not characteristic for the normal annual sales.

Calculated guesses estimate an order of magnitude of 200 tonnes of concentrate bought annually with 0.5-1.5% PFS, equivalent to 1.0-3.0 tonnes of PFS.

Emissions
The use of foam-forming concentrate for the extinction of fires obviously leads to the emission of PFS to the environment, dependent on the location. The collection of emissions from stationary systems is regulated. Most indoor locations are obliged to have a collection system for used fire-fighting foam. For many applications this is not possible; fire extinction will then lead to PFS emission (Ajax, 2002). Well-known examples are an accidental release at the International Airport of Toronto, Canada and use of AFFF on fire-fighting training sites (Moody & Field, 2000, Moody et al, 2002).
Since 2000, PFS containing fire-extinguishing agents are no longer used in Dutch military air force training sites (Koninklijke Luchtmacht, 2002).
AFFF concentrate has a long lifetime. If this lifetime has expired, the AFFF can be disposed as chemical waste, and is incinerated (Ajax, 2002). Incineration will presumably lead to the destruction of PFS.

A worst-case approximation would estimate the release to the environment of all the AFFF purchased. This would lead to the emission of 1-3 tonnes PFS annually.

3.3.6 Specialty surfactants
PFS are used as surfactants in various industrial applications. In total this group comprises a considerable amount of the PFS used, but it consists of various low volume applications. In the United Kingdom these applications accounted for 17.5% of the use of fluorinated chemicals as active ingredient (NCEHS, 2001). No general valid remarks can be made on the separate use figures and possible emissions.

3.3.7 Polymerisation aid
For the production of fluoropolymers, such as polytetrafluoroethylene (PTFE) a polymerisation aid is necessary. PFOA, or APFO (ammonium perfluoro-octanoate), improve physical properties of the polymer and increase the rate of polymerisation (Kissa, 2001).

Use figures
In the Netherlands only one production plant for fluoropolymers is present (Dupont, 2002). No data are available on the use in the Netherlands. Worldwide use is estimated to be 20 tonnes PFOA annually (Dupont, 2002).

Emissions
No data are available on the possible emissions from this application. Possible emissions occur from emissions from the plant and emissions from remaining PFOA in the finalised polymer. Industry is trying to reduce both, with considerable success (APME, 2002). Furthermore, the small amounts used make it unlikely that this application is a quantitatively important source of PFS to the environment (APME, 2002).

3.3.8 Production sites
In the Netherlands no PFS is produced. The nearest production plants are situated in Antwerp, Belgium (3M), and Villiers-St-Paul, France (formerly AtoFina, now Dupont (Atofina, 2002)). It is possible that emissions occur from these sites, resulting in the presence of PFS in the Netherlands, either by aerial or riverine transport.
Environmental monitoring has been executed upstream and downstream of a river to which the effluent of a fluorochemical production site is discharged in Decatur,

Alabama, USA (3M). This study pointed out that '*effluent from a fluorochemical manufacturing faculty may be one route of introduction in the environment of some environmentally prevalent organic fluorochemicals* (Hansen et al, 2002).' Moreover, preliminary results of an environmental monitoring study revealed elevated concentration of PFOS in aquatic organism downstream of a manufacturing plant in Antwerp, Belgium (Van de Vijver et al, 2002).

3.3.9 Other sources

Apart from other uses with corresponding possible emissions, one study revealed the formation of perfluorinated chemicals by thermolysis of fluoropolymers, such as PTFE. Possible products were longer chain polyfluoro (C3-C14) carboxylic acids (Ellis et al, 2001).
A major producer of fluoropolymers says that these results are very questionable. Their opinion has not been published (Dupont, 2002).

3.4 Summary of use figures

In table 3.6 the use figures from the last paragraphs are summarised. It becomes clear that paper & board is the most important application, followed by carpet, leather and presumably textile and specialty surfactants. For the last two no reliable data are available. Data from the UK suggest that these two applications take an important share of the PFS use.

Type of industry	Use of PFS (tonnes/year)	Form	Emissions (tonnes/year)
Carpet	15	Polymers	10 (worst case)
Paper & Board	*60-105* (not in NL)	Phosphates	
Textile	N.A.	Polymers	100% of the applied polymers
Leather	10-20	Polymers	
Fire-fighting foams (mobile)	0.13-0.81	Monomers	0.13-0.81 (worst case)
Fire-fighting foams (stationary)	1.0-3.0	Monomers	1.0-3.0 (worst case)
Specialty surfactants	N.A.	Monomers	-
Polymerisation aid	< 1	Monomers	-

Table 3.6. Use and emissions of PFS in the Netherlands. N.A. = not available

3.5 Overview of commercial names

All suppliers use different names for the same type of products. Not all suppliers offer products for all applications. This overview is not complete, but contains all major suppliers for the Dutch market.

Industry	3M	Atofina	Bayer	Ciba	Clariant	Dupont	Asahi glass	Daikin
Carpet	Scotchgard	Foraperle	Baygard	-		Zonyl	Asahiguard	Unidyne
Paper & board	Scotchban		Baysize-S, Baysynthol	Lodyne	Cartafluor			
Textile	FC brand textile		Baygard-K	Oleophobol	Pekophob			
AFFF	AFFF		-	-	-			
Leather	Scotchgard		Xeroderm	-	-			
Specialty surfactants	Various commercial names per suppliers							
Polymerisation aid	No commercial names							

Table 3.7. Commercial names of PFS products

3.6 References

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3M, 1999b, Material Safety data sheet of FC-603F 3M AR-AFFF 3%, St. Paul, Minnesota, United States of America

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3M, 2000d, Sulfonated perfluorochemicals: U.S. release estimation – 1997. Part 1: Life-cycle waste stream estimates, Final report, Battelle Memorial Institute, Columbus, Ohio, United States of America

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3M, 2002, personal communication with Dr. Sinnaeve, European toxicological manager, Antwerp, Belgium

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4 Behaviour in the aquatic environment

4.1 Introduction

The behaviour of organic micropollutants in the aquatic environment is determined by the properties of the compound (solubility, hydrophobicity, volatility) and by the characteristics of the water system of concern (residence time of the water, sedimentation area, organic matter content, etcetera). These compound and system properties determine to what extent a compound will accumulate in organisms.

Many data for perfluorinated surfactants are lacking, as was seen in the inventory of properties of perfluorinated surfactants in paragraph 2.2. Furthermore, it appears that some of the standard concepts of environmental modelling are not applicable for perfluorinated surfactants. This is explained in paragraph 4.3.

4.2 Solubility and volatilisation

The water solubility of a compound is a good indication of the extent to which the compound will be transported with water. In general, poorly soluble compounds have a high affinity for the organic matrix of silt particles in a water system.
Solubility and vapour pressure determine together whether a compound will evaporate out of the water. The volatility of a compound is characterised by its Henry constant. Since no Henry constants were available for the PFS, they have been calculated from the values for solubility and vapour pressure (Van Leeuwen & Hermens, 1995).

Substances for which no data were available are excluded.

Substance	Solubility (g/L)	P_{vapour} (Pa)	H (atm*m3*mol-1) (calc)
PFOS (K+)	0.519	0.331 mPa	3.4 E-9
PFBS	51	N.A.	-
PFOA (H+)	9.5	70	4.6 E-6
PFOA (NH4+)	> 500	< 1.3 mPa/ 9.2 mPa	<1.1 E-11/ 7.8 E-11
n-EtFOSE	151 µg/L	0.504	1.9 E-2
n-EtFOSEA	0.89 mg/L	N.A.	-
6:2 FTOH	12-17 mg/L	N.A.	~ 1 E-2
8:2 FTOH	140 µg/L	2.93	9.6 E-2

Table 4.1. Environmental relevant properties of selected fluorosurfactants

There is a large variation in solubilities, vapour pressures and Henry constants (see table 4.1). For PFOS the vapour pressure was determined to be 3.31 E-4 Pa. Although the vapour pressure determination study was rated with a Klimisch factor of 1, there is discussion about the reliability of the result (Cahill, 2002). The hydrogen-salt of PFOA is relatively volatile (70 Pa); the ammonium-salt is not (< 1.3 mPa) (Miteni, 2002). For PFOS and PFOA the combination of the good solubility, and their low vapour

pressure, resulting in low Henry constants, makes it unlikely that they will be transported by air over large distances (Renner, 2001, Martin et al, 2002).

N-EtFOSE, 6:2 FTOH and 8:2 FTOH have low solubilities. Combined with a moderately low vapour pressure, these chemicals have the tendency to escape from the water phase to air.

Martin and co-workers (2002) verified this suggestion in a preliminary study with only a few samples. They detected the presence in air of six fluorinated chemicals, of which at least three can be degraded (after deposition) into PFOS (see paragraph 4.4.2); these chemicals (n-EtFOSE, n-MeFOSE and n-EtFOSA) can thus play a role in the dissemination of perfluorinated chemicals in general and PFOS in particular (Martin et al, 2002). The other three chemicals were telomers; their degradation products are largely unknown hitherto (see, however, paragraph 4.4.3).
The results of this initial investigation by Martin and co-workers, combined with the volatility of some perfluorinated chemicals and the presence of PFOS in remote locations (Kannan et al, 2001a, Kannan et al, 2001b) indicate the potential of PFS to be transported over long distances.

4.3 Sorption

4.3.1 Octanol water partitioning

The distribution of a compound over n-octanol and water, commonly expressed by the partition coefficient K_{ow}, is often used to predict or mimic the partitioning between hydrophobic phases and water. K_{ow} has been proposed as a model for the partitioning between the body fat of biota and water (bioaccumulation), between the sediment and water (sorption) and to estimate the soil sorption coefficient for organic compounds (Sabljić et al, 1995).

This derivation of properties is based on the assumption that the hydrophobic and hydrophilic interactions between compound and substrate are the main mechanisms for the partitioning. This assumption has been shown to hold for non-polar and slightly polar organic chemicals.
There are two reasons why this concept is not applicable for fluorosurfactants. First, fluorosurfactants do not behave like traditional organic chemicals, due to the perfluorination: *'[..] hydrocarbon chains are oleophilic and hydrophobic, perfluorinated chains are both oleophobic and hydrophobic'* (Key et al, 1997). Therefore, PFS will not accumulate in fatty substances or adsorb to organic matter solely due to hydrophobic interactions. The oleophobic repulsion prevents the accumulation of PFS in fat (Kannnan et al, 2001a).
Second, fluorosurfactants are polar chemicals, intrinsically. PFOS is present in the environment as the dissociated salt (3M, 2001a) Therefore electrostatic interactions can play an important role in the distribution. Both biota and sediment have various polar parts with which interaction is very likely.

This 'theoretical' rejection of QSARs based on the octanol/ water partitioning is confirmed by the observation that PFS accumulate in blood plasma and liver, rather than in adipose tissue (Ylinen & Auriola, 1990, Olsen et al, 1999). PFOA is shown to bind to macromolecules in the tissue (USEPA, 2002). This is different from several persistent neutral lipophilic compounds (Kannan et al, 2001a), which accumulate in fat.

The suggestion that hydrophobic interactions are not the primary sorption mechanism is supported by the assumption that PFOS binds to sediment via chemisorption (3M, 2001b). Therefore, the K_{ow} is not suitable for the prediction of sorption of surfactants.

Although the significance of the partitioning between octanol and water is limited for environmental behaviour on PFS, there have been several studies that tried to determine the K_{ow} experimentally. Due to the surfactant properties of the substance it was not possible to obtain reliable results with the standard 'shake flask' method (3M, 2000a). Experiments with HPLC retention times made it possible to obtain more reliable K_{ow} results for n-EtFOSE and n-MeFOSEA (see table 4.2).

Substance	Log K_{ow}	References
n-EtFOSE	4.4	3M, 1994a
n-MeFOSEA	5.6	3M, 1994b

Table 4.2. Available values for partitioning n-octanol/ water

4.3.2 Sorption

The partitioning between sediment and water is an important factor in the fate of chemicals. Often, the portioning octanol/ water is used to predict this factor. In the previous section it was argued that this would not give correct predictions for perfluorinated compounds.

Direct measurements of the sorption to soil and sludge gave contradictory results for PFOS. Only two reliable studies are available (3M, 1978a, 3M, 2001b), with a Klimisch factor of respectively 2 and 1 (see annex 2). The first study predicts a high mobility of PFOS in Brill sandy loam soil (3M, 1978a). In the second study a strong adsorption to all soils tested was observed, including sludge and river sediment. Once adsorbed, PFOS does not desorb readily (3M, 2001b). The latter study suggests that the primary sorption process is chemisorption. In chemisorption the substance forms a chemical bond with the phase it is adsorbed to.

PFOA is reported to have a high mobility in Brill sandy loam soil, and it was suggested that PFOA (NH_4^+) has '[..] *the potential to migrate through soils to relatively shallow groundwater where it persists* (USEPA, 2002)'.
For other perfluorinated surfactants only less reliable study results are available, having a Klimisch factor of 3. Two studies suggest that n-EtFOSE is very likely to adsorb to soil (3M, 1978a, 3M, 1978b). For PFOA two totally contradictory conclusions were drawn from one study (3M, 1978c).
The results of a monitoring study near a fluorochemical plant show that PFOS, PFOA, FOSA and PFHS do occur in the sediment (3M, 2001c). These findings are supported by the detection of PFOS in various sediments and sewage sludge in a multi-city environmental monitoring study (3M, 2001d). Therefore it is unlikely that these fluorinated chemicals have a high mobility in sediment.

There are no data available on telomers. However, laboratory experiments show that 8:2 FTOH is rapidly sorbed from aqueous solutions. Specific recovery methods have been developed to be able to make accurate measurement. Experts state that the sorption of telomers onto various types of surfaces is very high and that desorption is very difficult (TRP, 2002).

4.4 Transformation

4.4.1 Introduction

The fluorine-carbon bond is the strongest single bond with carbon, but its strength is very much dependent on the actual molecular structure. Given this high energy, it is expected that many fluorinated organic compounds will be resistant to hydrolysis, photolysis and biodegradation (Smart, 1994). Indeed transformation rates (see table 4.3) suggest that PFS are relatively persistent in the environment.

The available data are collected in table 4.3.

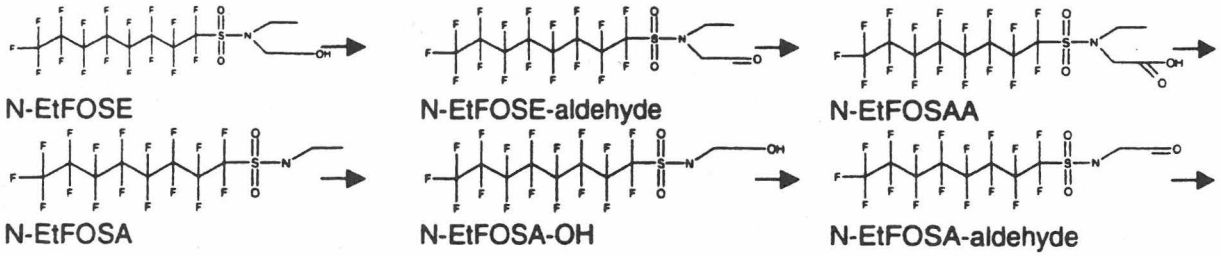
Substance	Biodegradation		Biotransformation	Photolysis	Hydrolysis
PFOS (K+)	0%[1,2,3,4,5,6]			0%[7,8]	T1/2[a] > 41 years[9]
PFOA (NH4+)	0%[2,10]			0%[11,12]	T1/2 > 92 years[13]
n-MeFOSE					T1/2 = 6.3 years[14]
n-EtFOSE	To PFOS/ PFOA[2,3]			0%[15]	T1/2 = 7.3 years[16] 92% after 24 hours to PFOS (alk
n-MeFOSEA					T1/2 = 99 days @ pH 7, 25°C (e
n-EtFOSEA					T1/2 35 days @ pH7, 25°C[19]
8:2 FTOH			To PFOA[20]	No direct photolysis expected[21]	

Table 4.3 Available data on the transformation of PFS, a) T1/2 = degradation half-life, (1) 3M, 1976 (2) 3M, 2001e (3) 3M, 2000b (4) 3M, 2000c (5) 3M, 2000d (6) 3M, 2000e (7) 3M, 1979a (8) 3M, 2001f (9) 3M, 2001g (10) 3M, 1978d (11) 3M, 1979b (12) 3M, 2001h (13) 3M, 2001i (14) 3M, 2001j (15) 3M, 1981 (16) 3M, 2001k (17) 3M, 1977 (18) 3M, 1999a (19) 3M, 1996 (20) Hagen et al, 1981 (21) TRP, 2002

4.4.2 ECF-products

Biodegradation

Many of the substances under study undergo primary degradation[2]. In this degradation step the non-fluorinated part of the molecule is transformed. The degradation pathway for n-EtFOSE in wastewater sludge is suggested to be as follows (3M, 2000b, 3M, 2001e):



[2] A compound is considered to be primary biodegradable if the original compound is altered, due to biodegradation processes. The degradation products can be persistent. With ultimate biodegradation, the original compound is completely transformed into CO_2 and H_2O and inorganic salts.

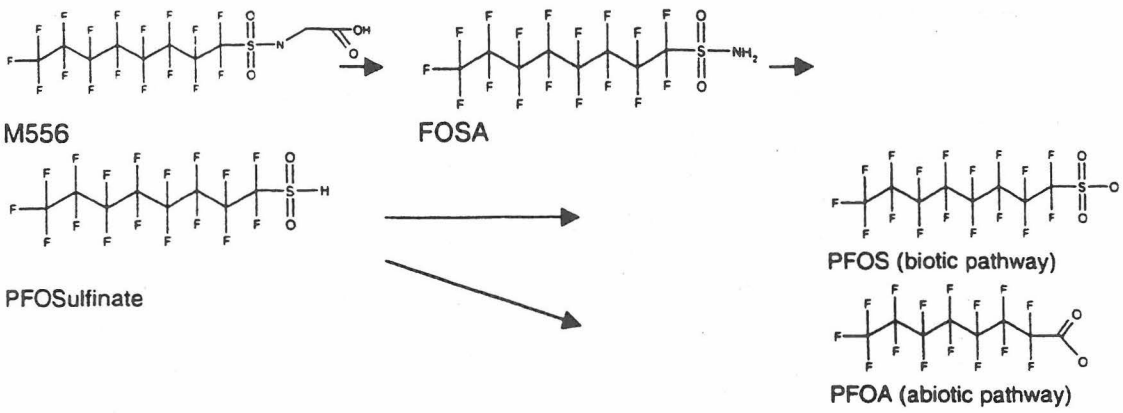


Figure 4.4. Degradation pathway of n-EtFOSE to PFOS and PFOA (3M, 2000b, 3M, 2001e).

It is likely that the degradation of n-MeFOSE will follow an analogous pathway, because n-MeFOSE contains the same reactive structures that appear to be vulnerable to microbial (biodegradation) attacks. N-EtFOSE and n-MeFOSE are the two main building blocks of the ECF-based fluorochemistry (3M, 2000h). No research has been published on the transformation of other functional groups, but it is expected that they can also be transformed to n-EtFOSE or n-MeFOSE. The likely endpoints of aerobic degradation of ECF-products are PFOS and PFOA (3M, 2001e). In both compounds the perfluoroalkyl chain is not affected by biodegradation. PFOS is non-degradable under both aerobic (3M, 1976, 3M, 2001e, 3M, 2000b, 3M, 2000c, 3M, 2000e) and anaerobic circumstances (3M, 2000d). PFOA is non-degradable under aerobic circumstances (3M, 1978d, 3M, 2001e); no anaerobic degradation test results are available.

Hydrolysis and photolysis

The available studies on photolysis show that this transformation mechanism will be of no importance in the breakdown of perfluorinated chemicals. The tests with PFOS, PFOA, POSF and n-EtFOSE show no photodegradation at all (see table 4.3).

Experiments show stability toward hydrolysis for all chemicals tested, with exception of the acrylates (see table 4.3). Both n-EtFOSEA and n-MeFOSEA are vulnerable to hydrolytic attack under environmental conditions. The transformation products are not known, but n-EtFOSE and n-MeFOSE, respectively, and acrylic acid are the most logical products. This transformation does not affect the perfluoroalkyl chain. Therefore, hydrolytic products of both acrylates will presumably not be affected by further hydrolysis, photolysis or biodegradation.

4.4.3 Telomer-products

Only one source dealing with the degradation of telomer-products in the environment is available. This study (Key et al, 1998) showed the degradation of 1H,1H,2H,2H-perfluorooctane sulfonate under sulfur limiting conditions by Pseudomonas sp. Strain D2. Volatile degradation products were formed, containing carbon, oxygen, hydrogen and fluorine. Furthermore, the detection of fluoride indicated defluorination (Key et al, 1998).

The biotransformation of a telomers has been investigated and published. This study revealed the biotransformation of 8:2 FTOH to PFOA in rats (Hagen et al, 1981). In this transformation two fluorine-carbon bonds are broken. Whether the same route of degradation is likely to occur in the environment is not known. If 8:2 FTOH is absorbed by biota, it is very likely that the same transformation will take place, leading to PFOA. In bioconcentration studies (see paragraph 4.5) it was shown that this chemical could be taken up by biota. Current research is dedicated to the biodegradation of telomer-products (Renner, 2001, TRP, 2002).
Although few experimental supporting data are available, there are various suggestions that the perfluoroalkyl chain of telomerisation products cannot be biodegraded, which is supported by the high binding energy of the fluorine-carbon bond (Smart, 1994, Key et al, 1997, Renner, 2001). However, the research by Key et al (1998) suggests that polyfluorinated alkyl chains are vulnerable to biodegradation, yielding biodegradation products that are different than those originating from ECF-products (PFOS, PFOA).
The Telomer Research Program (2002) concludes that direct photolytic degradation is not expected. A study on the indirect photolysis by OH radicals is underway.
The stability of the perfluoroalkyl chain makes it unlikely that it will be affected by photolysis.

4.4.4 Fluorinated polymers

The vast majority of the fluorochemicals are applied in polymeric form. Hence, most of emissions will be in (co-)polymeric form. Until now, no research has been done on the degradation or transformation of fluorinated polymers. This is an important subject, since, in general, polymers cannot cross membranes, and therefore will not have toxic effects. If monomeric PFS can be formed from polymeric fluorosurfactants, these could cross biotic membranes.
In interviews with manufacturers, it was suggested that fluorinated polymers are very stable (3m, 2002, Bayer, 2002). 3M states that they *'(..) have data demonstrating the stability of high molecular weight fluorochemical polymers and phosphate esters to various mechanisms of degradation.'* (3M, 2000f). One study, predicting the hydrolytic stability, is available. Although the data of this study have to be treated with caution, due to limited reliability, they show that fluoropolymers are rather stable to hydrolysis, resulting in half-lives ranging from 1-5 years for acrylates and esters to 500 years for fluorinated urethanes (3M, 2000g).
From a chemical point of view it seems possible to hydrolyse the ester bond in polyacrylates and polymethacrylates, leading to the formation of PFS. Also the ester bond in the fluoroalkyl phosphates might be vulnerable. These aspects have to be investigated in a reliable study.
An initial study with PFS treated textile has been performed. The organic extraction of polymer treated textile lead to the release of monomeric perfluorinated compounds. The origin of these monomers can be different than from transformation of the polymers (see paragraph 4.4.4) (Jonkers et al, 2002).

4.5 Bioconcentration

Bioaccumulation is a process in which a substance accumulates in an organism. There are two possible routes: biomagnification (uptake through food) and bioconcentration (uptake directly from the water).
Usually, for many organic compounds the bioaccumulation can be derived from the octanol/ water partitioning coefficient, because most organic chemicals accumulate in lipids. Since perfluorinated surfactants elicit a different partitioning behaviour, the K_{ow} is not a suitable predictor for the bioaccumulation (see paragraph 4.3.1).

Substance	Bioaccumulation	Bioconcentration	Biomagnification
PFOS (K+)	6300-125000[1]	484 (edible), 1124 (nonedible), 859 (whole) clearance >130d[2]	< 1[3]
PFOA (NH4+)		1.8[4] 2[3] <9.4[5] Clearance > 15d[6]	< 1[3]
8:2 FTOH		200-1100 (10 µg/L)[7] 87-310 (1µg/L)[7]	

Table 4.5. Available data on bioaccumulation, bioconcentration and biomagnification of PFS (1) Moody et al, 2002 (2) 3M, 20011 (3) Martin et al, 2001 (4) 3M in APME, 2002 (5) APME, 2002 (6) 3M, 1995 (7) METI cited in TRP, 2002

The available, reliable studies on bioaccumulation show that PFOS bioaccumulates, and is hardly excreted (see table 4.5). In an *in situ* bioaccumulation study in Canada a very high experimental bioaccumulation factor (BAF) for PFOS was observed: between 6300-125000 (Moody et al, 2002). This BAF is high in comparison with the BCF and BMF data available. Moody et al (2002) suggest that accumulated perfluorinated derivatives are metabolised to form PFOS, thus overestimating the BAF of PFOS.
In a laboratory experiment the BCF for PFOA (NH_4^+) was determined to be 1.8 for fish (fathead minnows), 2 for fish (Rainbow trout) and < 9.4 (Carp). The fathead minnows experiment is believed to have limited reliability (USEPA, 2002).

For other perfluorinated substances no bioaccumulation data are available.

4.6 Distribution

Research on the environmental fate of fluorinated chemicals is ongoing, including multi-species fate modelling (Cahill, 2002). Only one fate study using a fugacity model is available; this is qualified by the researcher as 'a small first step' (3M, 1999b). Although the preliminary fugacity modelling was tentative, and the Klimisch rating was 3, it is believed to give a rough approximation of PFOS behaviour (Cahill, 2002).
For this exercise the equilibrium criterion model (EQC) developed by Mackay et al (1996) was used. In this model there are three different levels, with increasing complexity (see figure 4.6).

This ECQ modelling predicts an 80/20 % partitioning over water and soil in level I and level II calculations. In level II, advection is the main removal mechanism. In level III calculations discharges to air and soil are predicted to partition to soil, whereas discharges to water are predicted to stay in the water and are subject to removal by adjective flow (3M, 1999b).

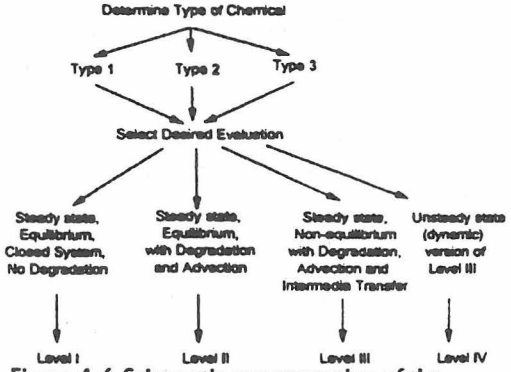


Figure 4.6 Schematic representation of the different levels in the EQC model (Mackay et al, 1996)

4.7 Conclusions and recommendations

From this chapter it became apparent that many data on the behaviour of PFS in the environment are not available. For telomers and fluorinated polymers, no data are available on sorption, degradation and distribution. It is recommended to fill these gaps of knowledge.

It became clear that n-EtFOSE, n-MeFOSE and n-EtFOSA (ECF products) and 6:2 FTOH, 8:2FTOH and 10:2 FTOH (telomers) can escape from the water phase to air, as they have been detected in air. For n-EtFOSE, 6:2 FTOH and 8:2 FTOH this tendency to leave the water phase is supported by relatively high Henry constants. These products can be transported over long range. It is very likely that these ECF products can degrade to form PFOS or PFOA. This mechanism can be an important factor in the global spreading of PFS.

The sorption of PFS cannot be modelled with Kow. Hydrophobic and hydrophilic interactions are not the primary partitioning mechanisms; presumably electrostatic interactions are. It is suggested that PFOS adsorbs via chemisorption. For PFOA no conclusions could be drawn considering the sorption potential.
Laboratory experiment show that 8:2 FTOH is rapidly adsorbed from aqueous solutions and desorption is very difficult. These preliminary results are supported by expert judgements.

The perfluoroalkyl chain of ECF products is not affected by degradation, photolysis or hydrolysis. The most likely end products of degradation are PFOS and PFOA. PFOS is not degraded under aerobic or anaerobic substances, for PFOA only aerobic results are available, showing the persistence of this substance. None of the tested chemicals can be transformed by light. Only the acrylates n-MeFOSEA and n-EtFOSEA can be transformed by hydrolysis, forming n-MeFOSE and n-EtFOSE, respectively, and acrylic acid.
The perfluoroalkyl chain of 6:2 FTOH is degraded in a study under sulfur limiting conditions, resulting in unidentified, volatile degradation products. In rats 8:2 FTOH is transformed to PFOA. 8:2 FTOH is not vulnerable to direct photolysis. There are various suggestions that the perfluoroalkyl chain of telomers cannot be (bio) degraded. No reliable data are available on the degradation or transformation of fluorinated polymers.

The bioaccumulation factor for PFOS is 6300-125000; the bioconcentration factor is 859 (whole fish). PFOA hardly bioconcentrates, with a BCF of 1.8 - < 9.4. The telomer alcohol 8:2 FTOH has a bioconcentration factor of 87- 1100.

When discharged to water, PFOS will partially adsorb to soil and sediment; bioaccumulation of PFOS will take place. Therefore, water, sediment and organic matter are believed to be the most important compartments.
PFOA will not evaporate from the water phase, and sorption is less, but it can persist in shallow groundwater. PFOA does not bioaccumulate. Therefore, water is believed to be the primary compartment for PFOA.

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5 Occurrence in the environment

5.1 Introduction

[redacted]

The perfluoroalkyl chain of fluorinated chemicals is persistent (see paragraph 4.4.1). Therefore they will be present in the environment. As was shown in chapter 4, their behaviour in the environment is not well known. It was shown that PFS accumulate in blood plasma and liver of biota. Various publications on the occurrence in the environment have been published. Most of these publications concern the occurrence in Northern American biota.
Very few data on the Western European situation are available and only the preliminary, non-reviewed results of one study on the occurrence in the Netherlands are available.

5.2 Analytical techniques (based on Giesy & Kannan, 2002)

5.2.1 Non-identifying methods

The fluorine content of organic molecules can be determined by destructive and nondestructive methods, such as neutron activation and X-ray fluorescence—low-sensitivity techniques that do not enable identification or quantification of individual organofluorine compounds.

Fluorine in organic compounds can also be determined by combustion, converting it to an inorganic fluoride; however, rigorous conditions are required for quantitative mineralisation. These techniques have been used for determining total fluorine in environmental and biological samples (Sweetser, 1965, Kissa, 1986). In environmental matrices, tests that measure methylene-blue-active substances have been used to detect anionic PFS, but the approach is non-specific (Levine et al, 1997).

5.2.2 GC-ECD/MS

Perfluorinated surfactants can be determined using derivatisation techniques coupled with gas chromatography followed by electron capture detection (Hagen et al, 1981) and mass spectrometric detection (Moody & Field, 1999, Moody & Field, 2000). PFOS has a low vapour pressure, and its derivatives are unstable.

5.2.3 HPLC-FD

Perfluorocarboxylic acid concentrations in biological samples have been measured using high-performance liquid chromatography (HPLC) and fluorescence detection (Ohya et al, 1998). Method application is limited to environmental samples.

5.2.4 NMR

Nuclear magnetic resonance (^{19}F NMR) can be used to determine perfluorinated surfactant concentrations in biological samples. NMR techniques have been used to measure PFS in contaminated water samples (Moody et al, 2001). The ^{19}F NMR-results were compared with LC/MS-data. It was suggested that the ^{19}F NMR technique overestimated the actual concentrations (see also section 5.3). In the 1970s, PFS in

human blood were analysed using non-quantitative NMR techniques (Hagen et al, 1981). Preconcentration is generally required with additional rigorous cleanup procedures.

5.2.5 HPLC/MS/MS

Compound-specific methods for analysing PFS using HPLC-negative ion electrospray tandem mass spectrometry (HPLC/MS/MS) (Hansen et al, 2001) enable surveys of the environmental distribution of PFS in wildlife at global scales (Giesy & Kannan, 2001, Kannan et al 2001a, Kannan et al, 2001b), but further method improvements are needed to accommodate the range of PFS concentrations in biological and environmental matrices and for monitoring PFS in atmospheric media.

5.3 Freshwater environment

PFOS, PFOA and FOSA have been analysed in a variety of media in six cities in the United States of America including drinking water, surface water column, sediment, publicly- owned treatment works (POTW) sludge, POTW effluent and landfill leachate samples. Decatur, Mobile, Colombus and Pensacola are so-called supply chain cities. In these cities perfluorinated chemicals are either manufactured or industrially used. Cleveland and Port St. Lucie are control cities. Results are listed below (table 5.1).

Sample	Decatur	Cleveland	Mobile	Columbus	Pensacola	Port St. Lucie
PFOS (parts per billion)						
POTW Effluent	4.98	0.436	0.048	0.427	0.896	0.069
POTW sludge(dry wt)	2980	123	58.9	158	125	61.6
Drinking water influent	N.D.	N.D	N.D.	0.057	N.D.	N.D.
Drinking water treated	N.D	N.D	N.D.	0.063	N.D.	N.D.
Drinking water tap	N.D	N.D	N.D.	0.058	0.045/N.D.	N.D.
Landfill leachate	52.7	N.C	N.D	N.D.	N.D.	0.382
Surface water	N.D/N.Q	N.D/N.Q	0.039	0.066	0.029/N.Q.	0.138/N.Q.
Sediment (dry wt)	0.452	N.D/N.Q	0.523	0.437	0.325/ N.D.	10.2
Quiet water	0.111	N.C	0.033	N.D.	N.Q.	2.19
Sample	Decatur	Cleveland	Mobile	Columbus	Pensacola	Port St. Lucie
PFOA (parts per billion)						
POTW Effluent	2.28	0.665	0.078	0.143	0.087	0.042
POTW sludge(dry wt)	173	0.297	N.Q.	16.4	2.46	N.D.
Drinking water influent	N.D.	N.D.	N.D.	0.026/N.Q.	N.D.	N.D.
Drinking water treated	N.D.	N.D.	N.D.	0.027	N.D.	N.D.
Drinking water tap	N.D	N.D.	N.D.	0.026/N.Q.	N.D.	N.D.
Landfill leachate	47.5	N.C.	N.D.	0.028/N.Q.	N.D.	0.946
Surface water	N.D./N.Q.	N.D.	0.056	0.026	N.D.	N.D
Sediment (dry wt)	N.D./N.Q.	N.D.	N.D./N.Q.	N.D.	N.D.	0.79
Quiet water	0.060	N.C.	0.027/N.Q	N.D.	N.D.	0.749
Sample	Decatur	Cleveland	Mobile	Columbus	Pensacola	Port St. Lucie

Sample	Decatur	Cleveland	Mobile	Columbus	Pensacola	Port St. Lucie
FOSA (parts per billion)						
POTW Effluent	0.056	N.Q.	N.Q.	0.085	N.Q.	N.Q.
POTW sludge(dry wt)	102.4	1.69	N.Q.	42.4	1.28	N.Q.
Drinking water influent	N.D.	N.D.	N.D.	N.Q.	N.D.	N.D.
Drinking water treated	N.D.	N.D.	N.D.	N.Q.	N.D.	N.D.
Drinking water tap	N.D.	N.D.	N.D.	N.Q.	N.D.	N.D.
Landfill leachate	0.254	N.C.	N.D.	N.D.	N.D.	N.Q.
Surface water	N.D.	N.D.	N.Q.	N.Q.	N.D.	N.D.
Sediment (dry wt)	N.Q.	N.D.	0.445	N.Q.	N.D.	N.Q./N.D.
Quiet water	N.Q.	N.C.	N.Q.	N.D.	N.D.	0.090

Table 5.1 PFOS, PFOA and FOSA in several media in six cities (average of duplicates; drinking water, surface water and sediment are averages of three different samples). N.D. = not detected, N.Q. = not quantifiable, N.C. = not collected (3M, 2001).

PFOS was detected most often, followed by PFOA and FOSA, all in relatively low concentrations. The highest concentrations were found in POTW sludge. The POTW effluent and landfill leachate were other important media (3M, 2001).
The highest concentrations were observed in Decatur. There is a fluorochemical manufacturing plant in this city. PFS was also present in the control cities, showing a general distribution of PFS.

The concentration of perfluorocarboxylates in groundwater near two airport fire-fighting training sites has been analysed (Moody & Field, 1999). The results listed in table 5.2 do not represent general groundwater concentrations.

Sample	N	PFHxA	PFHpA	PFOA	Total
Site 1.1	3	372	149	6570	7090
Site 1.2	5	57	18	460	540
Site 1.3	3	nd	Nd	nd	nd
Site 1.4	3	nd	Nd	nd	nd
Site 2.1	2	144	38	116	298
Site 2.2	2	73	22	64	159
Site 2.3	5	64	19	42	124
Site 2.4	2	nd	Nd	nd	nd

Table 5.2 Concentrations of perfluorocarboxylates in groundwater at two fire-fighting training sites (•g/L). nd = not detected above detection limit (Moody & Field, 1999).

Both sites showed contamination with PFS. Sites that were closer to the training-site were more heavily contaminated. PFOA is the quantitatively most important fluorochemical present.

The surface water concentrations of perfluorinated surfactants after an AFFF spill have been analysed with two different analytical methods (Moody et al, 2001, Moody et al, 2002). Results are listed in table 5.3.

Sample	Distance downstream from airport (km)	Total concentration (^{19}F NMR)	Total concentration (LC/MS/MS)	PFHxS	PFOS	PFOA
1-1	-3.9	N.D.	0.022	N.A.	N.A.	N.A.
1-2	4.1	3820	815	N.A.	N.A.	N.A.
1-3	6.6	4900	1090	N.A.	N.A.	N.A.
1-4	8.2	6000	1130	N.A.	N.A.	N.A.
1-6	15	N.D.	0.20	N.A.	N.A.	N.A.
2-1	-3.9	N.D.	0.011	N.D.	N.D.	0.011
2-2	4.1	311	93.5	3.45	N.D.	0.81
2-3	6.6	417	114	N.D.	89.2	0.61
2-4	8.2	539	133	5.44 (n=3)	126 (n=3)	1.60 (n=3)
2-5	9.7	900	185	8.22	174	2.49
2-6	15	17000 (n=3)	2270	49.6	2210	11.3
3-1	-3.9	N.D.	0.028	N.D.	N.D.	0.028
3-2	4.1	N.D.	1.92	N.A.	N.A.	N.A.
3-3	6.6	931	205	3.44 (n=3)	201 (n=3)	0.513 (n=3)
3-4	8.2	267	69.3	1.47	66.7	1.14
3-5	9.7	709	64.2	N.A.	N.A.	N.A.
3-6	15	N.D.	N.D.	N.A.	N.A.	N.A.

Table 5.3 Concentrations of PFS after an AFFF spill (•g/L). Sample 1-1 denotes the sample was collected 1 day after the spill at sampling site 1. N.D. = not detected, N.A. = not analysed. (Moody et al, 2001, Moody et al, 2002).

No PFS was detected upstream of the airport. The contamination is spread downstream over time. PFOS was the quantitatively most important fluorochemical present.

The surface water of a river upstream and downstream of a fluorochemical manufacturing facility in the USA has been analysed for perfluorinated surfactants. Both PFOS and PFOA levels increased downstream from the plant as can be seen in figure 5.4.

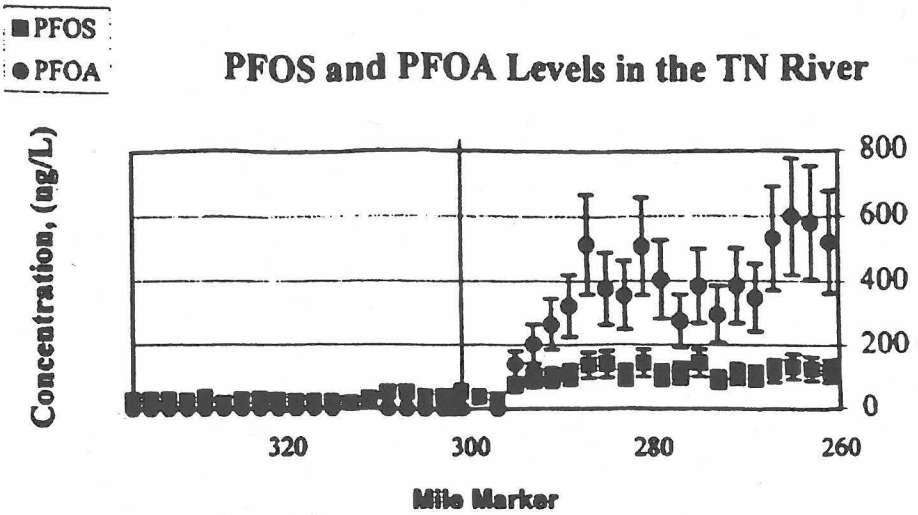

Figure 5.4 PFOS and PFOA Levels in the Tennessee River. The line at 301 Mile indicates the location of the incoming effluent from the fluorochemical manufacturing plant (Hansen et al, 2002).

The occurrence of telomers in the freshwater environment has not been studied.

5.4 Marine environment

No data are available on the occurrence of perfluorinated surfactants in the marine abiotic environment.

5.5 Biota

5.5.1 The Netherlands & Belgium

Until now only one study on the occurrence of PFS in the Dutch environment has been performed (Van de Vijver et al, 2002). This study revealed the presence of PFOS in various marine and estuarine organisms in the Western Scheldt estuary and the Belgian North Sea.

All samples that were analysed contained detectable amounts of PFOS. The highest average concentrations (> 1.7 •g/g tissue) were observed in plaice in the estuary (*Pleuronectus platessa*). Samples of shrimp and crab in the North Sea and the estuary showed concentrations between 40-300 ng/g tissue. Concentrations in *Trisopterus Luscus* (pouting) were the lowest: between 30 (North Sea) and 130 ng/g tissue (estuary) (Van de Vijver et al, 2002).

Presumably, these results are not representative for the entire Netherlands. Upstream of the Western Scheldt estuary, along the river Scheldt, a factory producing fluorochemicals is operating. Sampling near a fluorochemical plant in the United States showed that the plant is a possible source of emissions of PFS to the environment (see section 5.3) (Hansen et al, 2002). Therefore, concentrations downstream of the production site are expected to be higher than elsewhere.

There are no data available for telomers in biota from the Netherlands.

5.5.2 Europe

Giesy & Kannan (2001) and Kannan and co-workers (2001a) have published data on PFOS, in European seals, dolphins, cormorants and tuna. Results are shown in table 5.5. PFOS concentrations were well above detection limits and PFOS was found in all samples analysed. Concentrations were higher in the more urbanised areas. Only a few samples contained other PFS (FOSA, PFHS, PFOA) above the limit of quantification (LOQ) and have not been reported (Giesy & Kannan, 2001). Concentrations in individual organisms varied within about an order of magnitude.

At least one monitoring study is underway in Sweden. The preliminary results of that study showed low background concentrations in fish from unpolluted areas (1-2 ng/g fresh weight). Elevated PFOS levels were observed in fish from urbanised areas and a point source where fire-fighting foams had been used. Detailed results are not yet available (Järnberg, 2002).

Species	Location	Tissue	n	PFOS (ppm)
Ringed seal	Baltic Sea	Whole blood	29	158
Grey seal	Baltic Sea	Whole blood	26	38.3 (14-76)
Bottlenose dolphin	Mediterranean Sea	Liver	5	270 (170-430)
Striped dolphin	Mediterranean Sea	Liver	4	100 (65-160)
Common cormorant	Italy	Liver	12	96 (33-470)
Blue-fin tuna	Mediterranean Sea	Liver	8	48 (21-87)

Table 5.5 Concentrations of PFOS in European wildlife. Values in parentheses indicate range (Giesy & Kannan, 2001, Kannan et al, 2001a).

No data for the occurrence of telomers in biota in Europe are available.

5.5.3 Global occurrence

Several publications are available on the global occurrence of PFS in biota. Most information is available on concentrations of PFOS in wildlife from North America. In tables 5.6-5.12 levels observed in biota from North America and other parts of the world are presented.

As can be seen from table 5.6 shows large differences could be observed between individuals. Concentrations in eggs were higher than concentrations in liver and muscle.

Concentrations of PFOS in whole blood of birds were less than those in blood plasma (see table 5.7). Large differences between individual animals were observed. Concentrations of PFOS were much higher in species from more urbanised areas: PFOS concentrations are 10-100 fold less in species from the Midway Atoll than concentrations in species from the Mid Western USA.

In mustelids (table 5.8), invariably, PFOS was found above the LOQ. FOSA was the second most detected fluorinated chemical. Concentrations of PFOS in adults were higher than in juvenile mink. The suggested reason is a difference in feeding pattern (Kannan et al, 2002). Another possible explanation is the bioaccumulation potential of PFOS.
Concentrations of PFOS in mink and otter from more urbanised and industrialised areas were significantly higher than from more remote areas (Kannan et al, 2002).

In marine mammals (table 5.9) several patterns in PFOS concentrations could be observed. The most important explanations for differences in concentrations are location of feeding (concentrations higher closer to shore) and habitat (more remote locations give lower exposure) (Kannan et al, 2001a).
Within species a high variability in PFOS concentrations between individual organisms was observed.

Few samples of amphibians and reptiles have been analysed. From the data in table 5.10 it can be concluded that for turtles and frogs large differences in PFOS concentrations between individuals are possible.

The data presented in table 5.11 show that lower PFOS concentrations in biota from remote locations were considerably less than those observed from Europe and North America. Concentrations could not be quantified for many samples.
Large differences are observed between the same species from different locations: polar bear in the Beaufort Sea has tenfold lower concentrations on average than polar bear from several other locations (see table 5.11). Also ringed seal from Spitsbergen has a thirty fold lower concentration than ringed seal from the Baltic Sea.

Many more data are available from the United States of America than from the rest of the world. However, a comparison with the available data (see table 5.12) show that PFOS concentrations are highest in biota from North America, followed by biota from Europe. PFOS Concentrations in remote locations are much lower.

No data on the occurrence of telomers in biota are available.

Species	Location	Tissue	n	PFOS
Lake whitefish	Michigan waters	Eggs	2	260 (150-380)
Lake whitefish	Michigan waters	Liver	5	67 (33-81)
Lake whitefish	Michigan waters	Muscle	5	130 (97-170)
Brown trout	Michigan waters	Eggs	3	64 (49-75)
Brown trout	Michigan waters	Liver	10	<17-26
Brown trout	Michigan waters	Muscle	10	<6-46
Chinook salmon	Michigan waters	Liver	6	110 (33-170)
Chinook salmon	Michigan waters	Muscle	6	110 (7-190)
Carp	Saginaw Bay, Michigan	Muscle	10	120 (60-300)

Table 5.6 PFOS in fish from Northern America. Mean concentrations are given in ng/g wet wt for egg yolk, liver and muscle. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations (Giesy & Kannan, 2001).

Species	Location	Tissue	n	PFOS
Double Crested Cormorant		Whole blood	6	105 (34-188)
Cormorant	St. Matrin Is., Great Lakes	Whole blood	2	184 (124-243)
Double Crested Cormorant		Blood plasma	2	185 (63-372)
Double Crested Cormorant	Lake Winnipegosis, Manitoba, Canada	Egg Yolk	4	157 (21-220)

Species	Location	Tissue	n	PFOS
Double Crested cormorant	St. Martinville, LA	Liver	2	169 (51-288)
Herring Gull	Little Charity Is., Lake Huron	Whole blood	2	63 (57-68)
Herring Gull	Little Charity Is., Lake Huron	Blood plasma	2	315 (239-391)
Herring gull		Liver	5	186 (16-353)
Ring-Billed Gull	Sulphur Is., Thunder Bay, Lake Huron	Egg Yolk	3	67 (30-126)
Bald eagle		Blood plasma	33	320 (<1-2220)
Bald eagle		Liver	4	192 (24-467)
Black-crowned night heron	San Diego, CA	Liver	5	393 (32-648)
Brandt's cormorant	San Diego, CA	Liver	2	907 (46-1780)
Brown pelican		Liver	2	302 (118-533)
Common loon		Liver	14	129 (<12-595)
Franklin's gull	Red Rock Lakes, Beaverhead County, MT	Liver	4	40 (<12-61)
Great black-backed gull	Carteret County, NC	Liver	2	608 (187-841)
Great blue heron	St. Martinville, LA	Liver	2	539 (162-916)
Great egret		Liver	7	404 (27-1030)
Northern gannet	Carteret County, NC	Liver	1	85
Osprey		Liver	4	377 (42-959)
Red-throated loon		Liver	3	585 (34-1120)
Snowy egret		Liver	3	185 (43-413)
White pelican		Liver	6	270 (30-1120)
White-faced ibis	Sacramento Valley, CA	Liver	1	17
Wood stork	Charleston County, SC	Liver	1	158

Table 5.7 PFOS in piscivorous birds from North America. Mean concentrations are given in ng/mL for blood plasma and whole blood and in ng/g wet wt for egg yolk and livers. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations (Kannan et al, 2001b).

Species	Location	n	PFOS	FOSA	PFHxS	PFOA
Mink	Illinois	65	1177 (47-5140)	138	18	20
Mink	Massachusetts	31	298 (20-1100)	92	10	8
Mink	South Carolina	9	2081 (650-3110)	0	25	0
Mink	Louisiana	7	140 (40-320)	0	0	0
River Otter	Bremerton	1	288	22	<4	<7.5
River Otter	Eglon	2	297 (173-422)	60	<4	<7.5
River Otter	Fort Ward	3	156 (139-189)	55 (40-72)	<4-76	<7.5-19
River Otter	Silverdale	2	199 (151-248)	33 (27-39)	<4-52	<7.5-11

River Otter	Soleduck River	2	43 (25-62)	<4-4	<4	<7.5
River Otter	Willamette River	7	579 (97-994)	23 (4.4-44)	<4-68	<7.5-19
River Otter	Yaquina River	2	39 (34-45)	<4-7.4	<4	<7.5-9.9
River Otter	Nehalem River	1	82.8	13	<4	<7.5

Table 5.8 Concentrations of Perfluorochemicals in livers of Mink and otter in North America. Mean concentrations are given in ng/g wet wt. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations (Kannan et al, 2002).

Species	Location	Tissue	n	PFOS
Pygmy sperm whale		Liver	2	14.8 (6.6-23.0)
Short-snouted spinner dolphin	Gulf of Mexico	Liver	3	123 (78.7-168)
Striped dolphin		Liver	2	212 (36.6-388)
Rough-toothed dolphin		Liver	2	54.2 (42.8-65.6)
Bottlenose dolphin		Liver	20	489 (48.2-1520)
California sea lion		Liver	6	26.6 (4.6-49.4)
Elephant seal		Liver	5	9.3 (<5-9.8)
Harbor seal		Liver	3	27.1 (10.3-57.1)
Northern fur seal		Liver	5	329
Sea otter		Liver	8	8.9 (<5-14.3)
Sea otter		Brain	2	<35
Sea otter		Kidney	3	<35

Table 5.9 Concentrations of PFOS in Livers, kidney and brain of marine mammals in North America. Mean concentrations are given in ng/g wet wt. Values below LOQ are denoted by <. Means are calculated only for the detectable observations (Kannan et al, 2001a).

Species	Location	Tissue	N	PFOS
Yellow-blotched map turtle	Mississippi	Liver	6	190 (39-700)
Green frogs	Southwest Michigan	Liver	4	<35-290
Snapping turtle	Lake St. Clair, Michigan	Plasma	5	72 (1-170)

Table 5.10 Concentrations of PFOS in Liver and plasma of turtles and frogs from North America. Mean concentrations are given in ng/mL for blood plasma and in ng/g wet wt for liver. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations. (Giesy & Kannan, 2001)

Species	Locations	Tissue	N	PFOS
Weddel seal	Terra Nova Bay	Liver	1	<35
Polar skua	Terra Nova Bay	Plasma	2	<1-1.4
Black-footed albatross	Midway Atol, North Pacific Ocean	Liver	5	< 30

Species	Locations	Tissue	N	PFOS
Black-footed albatross	Midway Atol, North Pacific Ocean	Kidney	5	< 30
Black-footed albatross	Midway Atol, North Pacific Ocean	Serum	8	6.2 (3.0-17)
Laysan Albatross	Midway Atol, North Pacific Ocean	Liver	3	< 30
Laysan Albatross	Midway Atol, North Pacific Ocean	Kidney	3	<30
Laysan Albatross	Midway Atol, North Pacific Ocean	Serum	7	14 (5.7-34)
Yellow-fin tuna	Northern North Pacific ocean	Liver	12	<7
Northern fur seal	Pribilof Island	Liver	13	<10-122 [38][a]
Northern fur seal pup	Pribilof Island	Whole blood	19	<6-12 [5]
Northern fur seal adult	Pribilof Island	Whole blood	10	<6
Northern fur seal subadult	Pribilof Island	Whole blood	7	<6
Northern fur seal	Pribilof Island	Whole blood	8	<6
Polar bear	Beaufort Sea	Whole blood	14	34 (26-52)
Polar bear	Barrow; Nuiqsut; Point Lay; Gambell; Shishmaref; Little Diomede; Savoonga	Liver	17	350 (175-678)
Steller sea lion	Southeast Alaska	Whole blood	12	<6
Ringed seal	Spitsbergen	Whole blood	18	9.0
Ringed seal	Norwegian Arctic	Plasma	18	9 (5-14)
Gray seal	Sable Island	Whole blood	12	27.7 ± 11
Black-tailed gull	Korea	Liver	15	150 (70-500)
Black-tailed gull	Hokkaido, Japan	Plasma	24	6 (2-12)
Ganges river dolphin	Ganges River, India	Liver	2	<35-81

Table 5.11 Concentrations of PFOS in biota from locations outside North America and Europe. Mean concentrations are given in ng/mL for blood plasma and whole blood and in ng/g wet wt for liver and kidney. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations. Values in brackets [ ] indicate the percentage of detectable observations (Giesy & Kannan, 2001, Kannan et al, 2001a, Kannan et al, 2001b).

Species	Location	Tissue	n	PFOS (ppm)
Seal	North America	Liver	13	135 (<5-329)
Seal	Europe	Whole blood	55	101 (14-158)
Seal	Remote	Whole blood	64	16 (<6-38)
Dolphin	North America	Liver	27	396 (36.6-1520)
Dolphin	Europe	Liver	9	194 (65-430)
Cormorant	North America	Liver	4	538 (46-1780)
Cormorant	Europe	Liver	12	96 (33-470)
Tuna	Europe	Liver	8	48 (21-87)
Tuna	Remote	Liver	12	<7

Table 5.12 Comparison of PFOS concentrations between North America, Europe and remote concentrations. Mean concentrations are given in ng/mL for whole blood and in ng/g wet wt for liver. Values in parentheses indicate range. Values below LOQ are denoted by <. Means are calculated only for the detectable observations (Giesy & Kannan, 2001, Kannan et al, 2001a, Kannan et al, 2001b

5.6 Air

Martin et al (2002) have studied the occurrence of several perfluorinated surfactants in air. These authors detected fluorinated substances in samples collected at a highly urbanised site (Toronto) and a rural site (Long Point).

Substance	Toronto (n=4)	Long point (n=2)
	(pg^*m^{-3})	
N-MeFOSE	101	35
N-EtFOSE	205	76
N-EtFOSA	14 (n=2)	Not measured
4:2 FTOH	< LOD	< LOD
6:2 FTOH	87	29
8:2 FTOH	55	32
10:2 FTOH	29	17

Table 5.13 Concentrations of PFS in Canadian air samples (Martin et al, 2002). LOD = Limit of detection

Three ECF products and three telomers were detected and quantified in Toronto. Samples from the rural site showed considerably lower concentrations in air, but still five out of six attempted measurements demonstrated the occurrence of fluorinated chemicals in air.

5.7 Human exposure

Human exposure to organic fluorine has been observed as early as 1968. Taves (1968) concluded that '[..] *if in fact there is a non-exchangeable fluoride in serum, it did not break down or diffuse under these conditions, implying a large stable molecule. These findings are consistent with the presence of a fluorocarbon molecule.*'
With the development of analytical methods in recent years, the identification of organic fluorine compounds has improved. Although there has been some debate on the origin of organic fluorine in humans (Belisle, 1981), nowadays it is generally accepted that there is an anthropogenic origin.
Since 1993, several studies have been performed on the occurrence of PFS in humans. Olsen et al and Gilliland and Mandel published both two studies on levels of PFOS and PFOA in production workers with an occupational exposure (Gilliland & Mandel, 1993; Gilliland & Mandel, 1996; Olsen et al, 1999; Olsen et al, 2000). They found that PFOS and PFOA accumulated in human serum and liver.

PFOS and PFOA serum concentrations in occupationally exposed workers are in the 1-2 ppm range. Only the levels in workers from the Cottage Grove plant are higher.

In order to compare these data with the general population, also blood from people non occupationally exposed was analysed for PFOS and PFOA. Pooled serum samples from blood dated as far back as 1957 showed concentrations of several tens of ppb (OECD, 2002). Samples from 1998-2000 showed average serum levels between 17-53 ppb for PFOS and 3-17 ppb for PFOA. No differences could be observed between children (37.5 ppb) and elderly people (31 ppb).

Table 5.14 summarises the findings from these studies:

			PFOS		PFOA	
	Occupationally exposed					
Origin	Year	n	Mean (ppm)	Range (ppm)	Mean (ppm)	Range (ppm)
Cottage Grove Plant (USA)	1993	111	-	-	5.0	0.0-80.0
	1995	80	2.19	0.00-12.83	6.8	0.0-114.1
	1997	74	1.75	0.10-9.93	6.4	0.1-81.3
Decatur plant (USA)	1995	90	2.44	0.25-12.83	1.46	-
	1997	84	1.96	0.10-9.93	1.57	-
	1998	126	1.51	0.09-10.6	1.54	0.02-6.76
	2000	263	1.32	0.06-10.06	1.78	0.04-12.70
Antwerp plant (Belgium)	1995	93	1.93	0.10-9.93	1.13	0.00-13.2
	1997	65	1.48	0.1-4.8	-	-
	2000	258	0.80	0.04-6.24	0.84	0.01-7.04
Building 236 (USA)	2000	45	0.182	<0.037-1.036	0.106	0.008-0.668
Sagamihara (Japan)	1999	32	0.135	0.0475-0.628	-	-
	General population					
Origin	Year	n	Mean (ppb)	Range (ppb)	Mean (ppb)	Range (ppb)
Commercial sources (USA) (pooled)	1999	35	35	5-85	3	1-13
Blood banks (USA) (pooled)	1998	18	29.7	9-56	17[a)]	12-22
American Red Cross blood banks (USA)	2000	652	34.9	4.3-1656	5.6	4.27-52.3
Children (2-12y) (USA)	1999	599	37.5	6.7-515	5.6	4.27-56.1
3M Corporate managers (USA)	1998	31	47	28-96	12.5[b)]	Not reported
Plant management Sagamihara (Japan)	1999	32	40.3	31.9-56.6		
Plant management Tokyo (Japan)	1999	30	52.3	33-96.7		
Commercial sources, Intergen (USA)	1998	~ 500	44	43-44		
Commercial sources, Sigma (USA)	1998	~200	33	26-45		
Blood banks (the Netherlands) (pooled)	1999	5	53	39-61		
Blood banks (Belgium) (pooled)	1999	6	17	4.9-22.2		
Blood banks (Germany) (pooled)	1999	6	37	32-45.6		
Samples Seattle (65-96y) (USA)	1999	238	31	3.4-175		

Table 5.14 PFOS and PFOA serum concentration of production workers and general population (Olsen et al, 1999; Olsen et al, 2000, OECD, 2002, USEPA, 2002). A) PFOA detected in about 1/3 of the pooled samples but quantifiable in only two. B) Only 4 employees were above LOD of 10 ppb

5.8 Conclusions and recommendations

The data observed in the freshwater environment confirm that point sources of fluorochemicals lead to relatively higher levels of PFS in the nearby environment. Investigated point sources are a manufacturing plant, AFFF spills and industrial use. However, freshwater samples from cities that served as control site also contained PFS. Sewage sludge and to a lesser extent effluent and sediment are the most important media.

PFOS was detected in organisms around the globe, even in remote locations. Concentrations are higher in more urbanised or industrialised areas. Within species, sometimes, large differences are observed between individual organisms. All perfluorinated chemicals detected were present in liver, blood, muscle, kidney or brain. PFS does not bioaccumulate in the bladder.

A single study on the occurrence of PFS in the Dutch environment showed the presence of PFOS in several marine and estuarine biota. All available data on occurrence of PFOS in European biota show concentrations far above LOQ. Presumably PFOS can be transferred to offspring via the mother for birds.

PFOS concentrations in biota from North America exceed concentrations in biota from Europe. Concentrations in biota from remote regions were far lower.

No data are available for telomers in biota or water compartments.

Both ECF products and telomers have been detected in air in samples. Compared to an urban area, concentrations in samples from a more rural sampling site were considerably lower.

PFOS and PFOA have been detected in human blood samples. Concentrations in professionally exposed persons were about 50 (PFOS) to 250 times higher than concentrations in general public.

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6 Toxicity in the aquatic environment

6.1 Mechanism of toxicity

The mechanism of toxicity of PFS is not well understood. The perfluorocarboxylates (including PFOA) are peroxisome proliferators (Intrasuksri et al, 1998). Several other PFS are expected to exhibit the same mechanism of toxicity (Giesy & Kannan, 2002).

6.1.1 Metabolism

The scarcely available information on metabolism of shows that PFOS and PFOA are not transformed in biota (OECD, 2002, USEPA, 2002). 8:2 FTOH is transformed in rats into PFOA (Hagen et al, 1981).

6.2 Toxic effects in the aquatic environment

6.2.1 General

The aquatic toxicity and hazard to aquatic organisms of several PFS has been investigated in several studies. Thus, for the ECF products, many data are available. For the telomer products however, very few data are available. There are several reasons why the assessment of the aquatic toxicity of ECF products from these data is difficult (USEPA, 2002).

1) A variety of different lot numbers with different exact composition and impurities were tested. Impurities may affect toxicity. Moreover, the purity of the test material was not sufficiently tested. For some tests formulated products have been used, with varying concentrations of PFS. Other tests have been executed with impure chemicals, with as low as 19% of the test chemical present.
2) Testing occurred during a long period of time. During this period of time several different types of tests have been used, which makes the comparability of test results more difficult.
3) Water, isopropanol, or a combination of both were used with the test material in many of the toxicity tests, presumably as a carrier solvent. In tests where the test substance was not 100% pure, the toxicity values were corrected for the purity percentage.
4) In many of the tests only nominal test chemical concentrations were used. Measured test concentrations are always recommended, especially since it is known that PFS have a high sorption potential. Actual concentrations have indeed been observed that were significantly below nominal (OECD, 2002). Some tests have been performed at levels above the aqueous solubility. Results from these tests have not been included in the present evaluation.
5) For PFOS, tests have been performed with various counterions. It was assumed that the test results with different salts are comparable, since PFOS dissociates immediately to its anion and the according counterion. It is unlikely that these counterions are toxicologically significant, except for the dodecyldimethylammonium salt (DDA) (OECD, 2002).

In the present evaluation only studies that had a Klimisch value of 1 or 2 are included. The algae species *Selenastrum capricornutum* has been renamed *Pseudokirchneriella*

subcapitata (OECD, 2002). In this report the old name has been maintained. No tests results are available for sediment dwelling organisms. These organisms could be exposed to elevated concentrations in sediment.

6.2.2 Toxicity to freshwater organisms

Most of the reliable data that are available refer to PFOS and PFOA. Some reliable data are available for 8:2 FTOH, N-perfluorooctylsulfonyl-n-ethylglycinate (PFOSGE), N-EtFOSA, N-EtFOSE, N-EtFOSEA and POSF.

PFOS

Acute toxicity

In table 6.1 the freshwater acute toxicity values for PFOS are summarised. The data in table 6.1 show the moderate toxicity of PFOS to freshwater fish and invertebrates. The lowest reported EC_{50} is 4.7 mg/L for Fathead minnows. The lowest reported EC_{50} value for invertebrates was 27 mg/L for Daphnids. The test results for the PFOS-DDA salt show a much higher toxicity than for the potassium salt. As was discussed earlier, the didecyldimethylammonium salt may contribute to the overall toxicity. The values for mussel are in the same order of magnitude.

Algae are less sensitive to PFOS. Growth rate was used as end-point for the evaluation of toxicity to algae (USEPA, 2002). The lowest observed $EC_{50\ growth\ rate}$ for algae is 126 mg/L (96h). This is consistent with a 72h EC_{50} of 120 mg/L. For two other algae species the EC_{50}s were 176 and 305 mg/L, respectively.

The inhibition of leaf production for Duckweed was 108 mg/L (7d IC_{50}) with a NOEC of 15.1 mg/L.

Species	Protocol	Results (mg/L)	Comments	Ref
Fish				
Fathead Minnow	OECD 203	96h LC_{50} = 9.5 96h NOEC = 3.3	PFOS-K Measured concentration	1
	Not noted	96h LC_{50} = 4.7	PFOS-Li Extrapolated from 24,5% test substance	2
	OECD 203	96h LL_{50} = 200 96h NOEL = <170	PFOS-DDA Extrapolated from 35% test substance	3
Bluegill sunfish	OECD 203	96h LC_{50} = 7.8 96h NOEC = 4.5	PFOS-DEA Extrapolated from 25% test substance	4
Rainbow trout	Env. Canada	96h LC_{50} = 7.8	PFOS-K	5
	OECD 203	96h LC_{50} = 22	PFOS-K Measured concentrations	5
Invertebrates				
Daphnids	OECD 202	48h EC_{50} = 61 48h NOEC = 33	PFOS-K Measured concentrations	6
	ASTM/OECD 1981	48h EC_{50} = 27	PFOS-K	7
	Not noted	48h EC_{50} = 210 48h NOEC = 100	PFOS-Li Exposure is likely to be lower than nominal concentrations	8

	OECD 202	48h EL_{50} = 4.0 48h NOEL = 2.2	PFOS-DDA Extrapolated from 35% test substance	9
	ISO, 1982	48h EC_{50} = 58	PFOS-K	5
Freshwater mussel	OECD 203	96h LC_{50} = 59 96h NOEC = 20	PFOS-K	10
Algae				
Selenastrum Capricornutum	OECD 201	96h $EC_{50\ (cell\ density)}$ = 71 96h $EC_{50\ (area\ inder\ the\ curve)}$ = 71 96h $EC_{50\ (growth\ rate)}$ = 126 96h NOEC $_{(cell\ density,\ area\ under\ the\ curve,\ growth\ rate)}$ = 44 72h $EC_{50\ (cell\ density)}$ = 70 72h $EC_{50\ (area\ inder\ the\ curve)}$ = 74 72h $EC_{50\ (growth\ rate)}$ = 120 72h NOEC $_{(cell\ density,\ area\ under\ the\ curve,\ growth\ rate)}$ = 70	PFOS-K	11
	OECD 201	96h $EC_{50\ (cell\ density)}$ = 82 96h $EC_{10\ (cell\ density)}$ = 10	PFOS-K	12
	OECD 201	96h $EC_{50,\ cell\ dry\ weight}$ = 115 96h $EC_{50,\ cell\text{-}count}$ = 82	PFOS-K	13
Anabaena flos-aquae	OPPTS 850.5400	96h $EC_{50\ (growth\ rate)}$ = 176 96h $NOEC_{(growth\ rate)}$ = 94	PFOS-K Measured concentrations	12
Navicula pelliculosa	OPPTS 850.5400	96h $EC_{50\ (growth\ rate)}$ = 305 96h $NOEC_{(growth\ rate)}$ = 206	PFOS-K Measured concentrations	14
Higher plants				
Duckweed	OPPTS 850.4400	7d IC_{50} = 108 7d NOEC = 15.1 mg/L	PFOS-K Measured concentrations	15

Table 6.1 Acute toxicity of PFOS to freshwater organisms.

Sub-chronic/ chronic toxicity

Fish appear to be much more sensitive than invertebrates and algae to sub-chronic/ chronic exposure to PFOS (see table 6.2). The same pattern was found with acute toxicity. The NOEC of 0.30 mg/L is consistent with results from a bioconcentration study. In that study no effects were measured at 0.086 mg/L during 62 days uptake, but 100% mortality occurred at an exposure concentration of 0.87 mg/L during 35 days.

The two available studies for daphnids show consistent results.

Species	Protocol	Results (mg/L)	Comments	Ref
Fish				

Fathead Minnow	OECD 210	42d $NOEC_{surv}$ = 0.30 42d $NOEC_{growth}$ = 0.30 5d $NOEC_{hatch}$ => 4.6	PFOS-K Measured concentrations	16
	Non-standard	30d $NOEC_{early\ life\text{-}stages}$ = 1		17
Bluegill sunfish	OECD 305	62d $NOEC_{mortality}$ > 0.086 <0.87	PFOS-K Bioconcentration study Measured concentrations	18
Invertebrates				
Daphnids	OECD 211	21d $NOEC_{repro}$ = 12 21d $NOEC_{surv}$ = 12 21d $NOEC_{growth}$ = 12	PFOS-K Measured concentrations	19
	ASTM/OECD, 1981	21d $EC_{50,\ repro}$ = 12 28d $NOEC_{repro}$ = 7 28d $EC_{50,\ repro}$ = 11	PFOS-K	7

Table 6.2. Sub-chronic/chronic toxicity of PFOS to freshwater organisms

PFOA

Acute and sub-chronic/ chronic toxicity

In table 6.3 the acute and (sub-) chronic freshwater toxicity values for PFOA are summarised. Only studies that had a Klimisch value of 1 or 2 are reported. In the draft hazard assessment of the USEPA (2002) several other ecotoxicity data are reported. However, the reliability of some of these studies was limited. Only studies for which the reliability could be assessed were included in the present review.

The test results of acute toxicity of PFOA to freshwater species show a wide variation. For fish LC_{50} values vary between 300-766 mg/L. These results indicate low acute toxicity of PFOA to fish.
For Daphnids the EC_{50} range observed is 15-720 mg/L. The range corresponds to a moderate to low toxicity.
For algae the $EC_{50\ growth\ rate}$ values range between 3.8 and > 1000. The lower value indicates high to moderate toxicity, the high value indicates very low toxicity.
To bacteria and activated sludge, PFOA exhibits low toxicity.

The few available sub-chronic/ chronic values for PFOA indicate a relatively low toxicity of this compound.

Species	Protocol	Results (mg/L)	Comments	Ref
Fish				
Fathead Minnow	USEPA 660/3	96h LC_{50} = 766 96h LC_{50} = 400		20
	Not noted	96h LC_{50} = 300	Extrapolated from 78-93% test substance	21
	OECD 203	96h LC_{50} > 450	Extrapolated from 45% test substance	22
	EPA/TSCA 1993	96h LC_{50} = 494	Extrapolated from 20% test substance	23
	Not noted	96h LC_{50} = 843		24

Species	Protocol	Results (mg/L)	Comments	Ref
	EPA/TSCA 797.1400	96h LC_{50} = 432 96h NOEC = 284	Extrapolated from 45% test substance	25
	EPA/TSCA 797.1050	96h NOEC = 400 96h NOEC = 270	Extrapolated from 45% test substance	26
	OECD 202	48h EC_{50} = 263	Extrapolated from 45% test substance	27
	EPA/TSCA 797.1300	48h EC_{50} = 240 48h NOEC = 146	Extrapolated from 20% test substance	28
	EPA/TSCA 797.1300	48h EC_{50} = 720 48h LC_{50} = 720 48h NOEC = 360		29
	EPA/TSCA 797.1300	48h EC_{50} = 15 48h LC_{50} = 35 48h NOEC = 6	Extrapolated from 45% test substance	30
Algae				
Selenastrum Capricornutum	EPA/TSCA 797.1050	96h EC_{50} = 2.2 96h NOEC = 0.45	Extrapolated from 45% test substance	31
	EPA/TSCA 797.1050	96h $EC_{50,\ cell\ density}$ = 1.3 96h $EC_{50,\ growth\ rate}$ = 3.8	Extrapolated from 45% test substance	32
	OECD 201/ EPA/TSCA 797.1050	96h EC_{50} = 396 96h $EC_{50,\ growth\ rate}$ = 666 96h $NOEC_{cell\ count}$ = 42 96h $NOEC_{growth\ rate}$ = 86 96h $LOEC_{cell\ count}$ = 86 96h $LOEC_{growth\ rate}$ = 166	Extrapolated from 20% test substance	33
	EPA/TSCA 797.1050	96h $EC_{50,\ cell\ density}$ = 310 96h $EC_{50,\ growth\ rate}$ > 1000 96h $NOEC_{cell\ density}$ = 62 96h $NOEC_{growth\ rate}$ = 500		34
Bacteria				
Photobacterium phosphoreum	Microbics microtox	30min EC_{50} = 722	Extrapolated from 83% test substance	35
	Microbics microtox	30min EC_{50} >450	Extrapolated from 45% test substance	36
	Microbics microtox	30min EC_{50} = 730		37
	Microbics microtox	30min EC_{50} = 630	Extrapolated from 20% test substance	38
	Microbics microtox	30min EC_{50} = 390	Extrapolated from 20% test substance	39
	Microbics microtox	30min EC_{50} = 117	Extrapolated from 45% test substance	40
Activated sludge				
	OECD 209	3h EC_{50} > 450	Extrapolated from 45% test substance	41
	OECD 209	3h EC_{50} > 664	Extrapolated from 20% test substance	42
	OECD 209	3h EC_{50} > 450	Extrapolated from 45% test substance	43

Species	Protocol	Results (mg/L)	Comments	Ref
Fish- chronic				
Fathead minnows	Adapted EPA, 1972	30d NOEC > 100		44
Algae- chronic				
Selenastrum Capricornutum	Modified EPA/ASTM/OECD	14d $EC_{50,\ cell\ count}$ = 43		45

Table 6.3 Toxicity of PFOA to freshwater organisms.

8:2 FTOH

Acute toxicity

No effects have been observed in toxicity tests with 8:2 FTOH (see table 6.4). Test results were based on nominal concentrations. It is not possible to appropriately judge the toxicity of this telomer from these data.

	Species	Protocol	Results (mg/L)	Comments	Ref
Fish	Danio rerio	OECD 203	96h NOEC = 0.18	No effects at limit of solubility	46
Invertebrates	Daphnids	OECD 202	48h NOEC = 0.16	No effects at limit of solubility	46
Algae	Scenedesmus subspicatus	OECD 201	72h NOEC = 0.20	No effects at limit of solubility	46
Activated sludge		OECD 209	3h NOEC > 1000	Highest concentration tested	46

Table 6.4 Acute freshwater toxicity of 8:2 FTOH to freshwater organisms

Other PFS

For the remaining PFS discussed in the present study the toxicity data set is far from complete (see table 6.5). For PFOSGE L(E)C_{50}s are available for the three trophic levels (Fish, Daphnia, Algae). Daphnia are the most sensitive organisms with an EC_{50} = 0.29 mg/L, followed by fish (LC_{50} = 11). PFOSGE exhibits low toxicity to algae.

N-EtFOSA is moderately toxic to daphnia (EL_{50} = 14.5 mg/L) and has a low toxicity to fish.

PFDS exhibits moderate toxicity to fish and daphnids, with an LC_{50} of 4.8 and 11 mg/L, respectively.

	Species	Protocol	Results (mg/L)	Comments	Ref
PFOSGE					
Fish	Fathead Minnow	OECD 203	96h LC_{50} = 11 96h NOEC < 1.9	Extrapolated from 19% test substance	47
		OECD 203	96h LC_{50} = 41 96h NOEC = 23	Extrapolated from 42% test substance	48
		EPA	96h LC_{50} = 362	Extrapolated from 42% test substance	49
Invertebrates	Daphnids	OECD 202	48h EC_{50} 0.29 48h NOEC = 0.19	Extrapolated from 19% test substance	50

	Species	Protocol	Results (mg/L)	Comments	Ref
Algae	Selenastrum Capricornutum	OECD 201	96h $EC_{50,\ cell\ count}$ = 125 96h $EC_{50,\ growth\ rate}$ =254 96h NOEC = 91	Extrapolated from 42% test substance	51
Bacteria	Photobacterium phosphoreum	Microbics microtox	30min EC_{50} = 78	Extrapolated from 19% test substance	52
		Microbics microtox	30m EC_{50} = 115	Extrapolated from 42% test substance	53
Activated sludge		OECD 209	3h EC_{50} > 190	Extrapolated from 19% test substance Highest concentration tested	54
N-EtFOSE					
Fish-chronic	Fathead minnow	USEPA, 1972	NOEC > 20 µg/L	Highest concentration tested	55
N-EtFOSEA					
Fish	Fathead minnow	Not noted	96h LC_{50} > 1000	Highest concentration tested	56
POSF					
Fish	Fathead minnow	Not noted	96h LC_{50} > 1000	Highest concentration tested	57
N-EtFOSA					
Fish	Fathead minnows	Adapted OECD 203	96h LL_{50} = 206		58
Invertebrates	Daphnids	Adapted OECD 202	48h EL_{50} = 14.5		59
Activated Sludge		OECD 209	3h EC_{50} > 1000	Highest concentration tested	60
PFDS					
Fish	Fathead minnows	OECD 203	96h LC_{50} = 4.8	Extrapolated from 25% test substance	61
Invertebrates	Daphnids	OECD 202	48h EC_{50} = 11	Extrapolated from 25% test substance	62
		EPA 660/3	48h EC_{50} = 32	Extrapolated from 25% test substance	63
Bacteria	Photobacterium phosphoreum	OECD 209	17.3% inhibitory at 250 mg/L	Extrapolated from 25% test substance Highest concentration tested	64
Activated sludge		Microbics microtox	30min EC_{50} = 327	Extrapolated from 25% test substance	65

Table 6.5 Acute and chronic toxicity data of several PFS to freshwater organisms.

6.2.3 Summary of freshwater toxicity data

The lowest effect concentrations and NOECs that have been published in the literature have been summarised in table 6.6.

Substance	Acute/chronic	Trophic level	Species	Results (mg/L)
PFOS	Acute	Fish	Fathead minnow	96h EC_{50} = 4.7
		Invertebrates	Daphnids	48h EC_{50} = 27
		Algae	Selenastrum Capricornutum	96h $EC_{50,\ growth\ rate}$ = 126
	Chronic	Fish	Fathead minnow	42d NOEC = 0.30
		Invertebrates	Daphnids	28d $NOEC_{reproduction}$ = 7
PFOA	Acute	Fish	Fathead minnow	96h LC_{50} = 300
		Invertebrates	Daphnids	48h LC_{50} = 15
		Algae	Selenastrum Capricornutum	96h $EC_{50,\ growth\ rate}$ = 3.8
8:2 FTOH	Acute	Fish	Danio rerio	96h NOEC = 0.18
		Invertebrates	Daphnids	48h NOEC = 0.16
		Algae	Scenedesmus subspicatus	72h NOEC = 0.20
PFOSGE	Acute	Fish	Fathead minnow	96h LC_{50} = 11
		Invertebrates	Daphnids	48h EC_{50} = 0.29
		Algae	Selenastrum Capricornutum	96h EC_{50} = 254
N-EtFOSA	Acute	Fish	Fathead minnow	96h LL_{50} = 206
		Invertebrates	Daphnids	48h EL_{50} = 14.5
PFDS	Acute	Fish	Fathead minnow	96h LC_{50} = 4.8
		Invertebrates	Daphnids	48h EC_{50} = 11

Table 6.6 Lowest observed $L(E)C_{50}$ and NOECs of PFS in freshwater organisms.

6.2.4 Toxic effects in the marine environment

For the marine environment toxicity data are only available for PFOS. Table 6.7 presents the published toxicity data for marine organisms.

The few data that are available for the toxicity of PFOS to marine organisms show moderate toxicity to invertebrates. For fish no conclusions can be drawn, due to the limited reliability of the rainbow trout study. The test with algae did not show any effects at the highest concentration tested.
In a chronic study with shrimps NOECs between 0.25-0.55 mg/L were derived.

Species	Protocol	Results (mg/L)	Comments	Ref
Fish				
Sheepshead minnow	OECD 203	96h LC_{50} > 15 mg/L	PFOS-K Measured concentration	66
Rainbow trout	Env. Canada	96h LC_{50} = 13.7	PFOS-K Possibly tested in concentration higher than salt water solubility	5
Invertebrates				

Species	Protocol	Results (mg/L)	Comments	Ref
Mysid shrimp	OPPTS 850.1035	96h LC_{50} = 3.6 96h NOEC = 1.1	PFOS-K Measured concentration	67
Eastern Oyster	OPPTS 850.1025	96h EC_{50} > 3.0 96h NOEC = 1.9	PFOS-K Measured concentration	68
Algae				
Skeletonema costatum	OPPTS 850.5400	96h $EC_{50\ (growth\ rate)}$ => 3.2 96h $NOEC_{(growth\ rate)}$ => 3.2	PFOS-K Measured concentration Highest concentration tested	69
Invertebrates- chronic				
Mysid Shrimp	OPPTS 850.1350	35d $NOEC_{repro}$ = 0.25 35d $NOEC_{surv}$ = 0.55 35d $NOEC_{growth}$ = 0.25	PFOS-K Measured concentrations	70

Table 6.7 Toxicity of PFOS to marine organisms.

6.3 Standards and derivation of IMPCs (Based on Groshart et al, 2001)

6.3.1 Introduction

In the Netherlands, harmonised standards for several environmental compartments are derived for a number of chemicals (MilBoWa, 1999). The purpose of MilBoWa (1999) is to create a system of limit- and target values for soil and surface water.
A limit value is a quality level that minimally should be achieved or maintained. A target value is a quality level at which no adverse effects are expected. The limit value is based upon the 'maximal permissible concentration' (MPC), the target value on the 'negligible concentration' (NC). Previously different MPCs were operative for the same substance because there were also MPCs derived in the framework of the admission of plant protection products and biocides. In 1999 (Kalf, et al, 1999) the procedure for the derivation of MPCs for admission policy of plant protection products and biocides and the setting of environmental quality standards were harmonised.
The MPC is defined as the concentration at which at least 95% of the species in the ecosystem will be protected (method of Van Straalen and Denneman (1989), modified to the model of Aldenberg and Slob (1991; 1993). The negligible risk level is defined as 1% of the MPC.

For PFS there are no standards derived yet in the Netherlands.

6.3.2 Derivation method

For the derivation of MPCs directly from ecotoxicological endpoints two different methods are used: the refined effect assessment method and the preliminary effect assessment method. Because long-term chronic data are preferred above short term acute data the aim is to apply the refined effect assessment method. However application of this method is based on data availability: at least four NOEC values are needed for four different taxonomic groups of organisms. If these data are not available the preliminary effect assessment method is applied. In this case in principle the TGD is applied. In figure 6.8 the direct method for MPC derivation is presented.

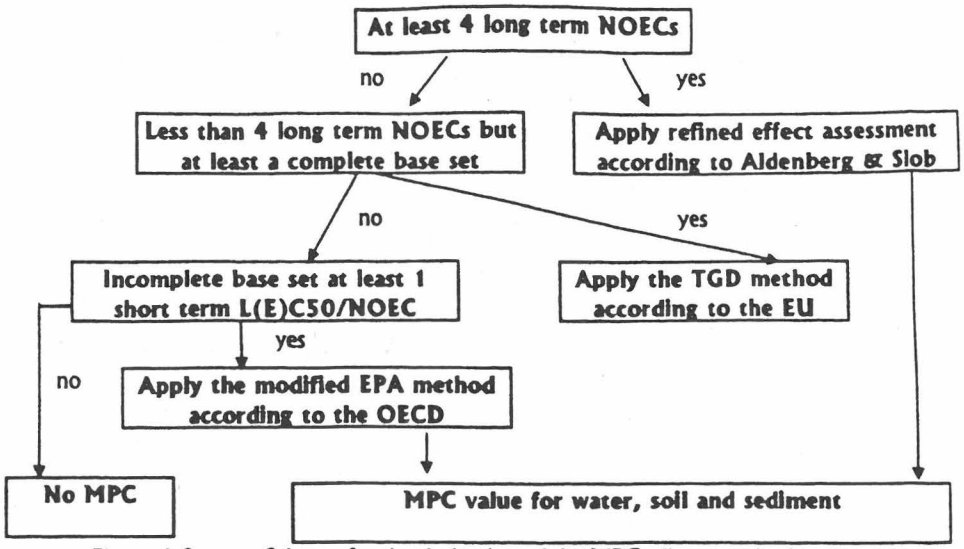

Figure 6.8 Scheme for the derivation of the MPC: direct method

Available valid data	Assessment factor to be applied to the lowest L(E)C50 or long-term NOEC
At least one short-term L(E)C50 from each of 3 trophic levels of the base-set (fish, Daphnia and algae)	1000
One long-term NOEC (either fish or Daphnia)	100
Two long-term NOECs from species representing two trophic levels (fish and/or Daphnia and/or algae)	100 50
Long-term NOECs from at least 3 species (normally fish, Daphnia and algae) representing three trophic levels	50 10
Field data or model ecosystems	Reviewed on a case by case basis

Table 6.9 Assessment factors for aquatic toxicity data following EU/TDG (ECB, 1996) according to EUSES (EC, 1996)

There are two exceptions to the use of the TGD method:

1. Only when long term NOECs on three trophic levels are available, a comparison with data from the (complete) base set is no longer demanded.
2. It is inferred that for more hydrophobic compounds, short term toxicity data may not be representative, since the time span of an acute test may be too short to reach a toxic internal level. In those cases, base set completeness is not demanded and an assessment factor of 100 may be applied to a chronic test, which should not be an alga test if this is the only chronic test available.

If the base set is incomplete, the TGD method cannot be applied, arbitrary safety factors are used (the modified EPA-method (OECD, 1992)): a factor 10 and/or 1000 will be applied to the NOEC and/or L(E)C50, respectively, to derive the MPC. It should be stressed here that this exception may only be used if the TGD can not be applied.
In table 6.10 the safety factors of the modified EPA method, dependent on the number of available toxicity data, are presented.

The calculated MPC in this report will be defined as 'indicative MPC' (iMPC).
In contradiction to the limit and target values the derived iMPCs have only a technical status and no political value. They are not legally set and may change as soon as more toxicity data become available and/or an MPC is derived by the INS-project.

Available toxicity data	Safety factor
Lowest acute L(E)C_{50} or QSAR estimation for acute toxicity	1000
Lowest acute L(E)C_{50} or QSAR estimation for acute toxicity for at least algae, crustaceans and fish	100
Lowest NOEC or QSAR estimation for chronic toxicity	10*
Lowest NOEC or QSAR estimation for chronic toxicity for at least algae, crustaceans and fish	10

Table 6.10 Safety factors for the derivation of iMPCs in surface water (modified EPA method)

* this value will be compared with the value based on acute L(E)C_{50} values. The lowest value will be selected

Based on the toxicity data that were presented in this study, the iMPCs are derived using the procedure described by Kalf (1999). The derivation is explained in annex VI.
To derive the iMPC for sediment it is advised to use the equilibrium partition (EP) method (see Slooff, 1992; Beek, 1993; Kalf, 1999). In this study this advice has not been followed, because K_{ow} is not a suitable predictor for the environmental behaviour (see paragraph 4.3.1). Furthermore, the $iMPC_{sediment}$ could not be derived direct from effect concentrations, because no data were available on toxic effects in the soil or sediment.
In the present study iMPCs for PFOS, PFOA and PFOSGE were derived. For all other PFS insufficient data were available. The results of this derivation are presented in table 6.11

Substance	$iMPC_{freshwater}$ (µg/L)	$iMPC_{marine\ water}$ (µg/L)
PFOS	6	1.1
PFOA	3.8	-
PFOSGE	0.29	-

Table 6.11 iMPCs for PFOS, PFOA and PFOSGE.

The differences in iMPCs are mainly due to differences in data availability. Only for PFOS many data were available, making it possible to use a small assessment factor. All other data have been derived using an assessment factor of 1000 (see annex VI).

6.3.3 Comparison of iMPCs to environmental concentrations

The PFS concentrations that are observed in the environment can be compared to the indicative MPC. No occurrence data are available for PFOSGE, therefore this comparison will be limited to PFOS and PFOA.

PFOS

The highest freshwater concentrations that were observed in the multi-city environmental monitoring study (see paragraph 5.3) were 4.98 •g/L for PFOS in POTW effluent from Decatur. In quiet water from Decatur 0.111 •g/L PFOS was observed. The highest PFOS water concentration from control cities is 2.19 •g/L (Port St. Lucie). The highest PFOS water concentration after an AFFF spill (see paragraph 5.3) was 2210 •g/L.

These values indicate that the iMPC for PFOS can be exceeded due to point sources of PFS. However, also PFOS concentrations in a non-point sourced city could approach the iMPC.

PFOA

The highest freshwater concentrations that were observed in the multi-city environmental monitoring study (see paragraph 5.3) were 2.28 •g/L for PFOA in POTW effluent from Decatur. In quiet water from Decatur 0.060 •g/L PFOA was observed. The highest PFOA water concentration from control cities is 0.749 •g/L (Port St. Lucie). The highest PFOA water concentration in groundwater at a fire-fighting training site is 6570 •g/L; after an AFFF spill the highest observed PFOA concentration was 11.3 •g/L. These values indicate that the iMPC for PFOA can be exceeded due to point sources of PFS.

6.4 Human toxicity

The human toxicity of PFOA and to a lesser extent PFOS have been and still are the subject of many studies (USEPA, 2002, OECD, 2002). For 8:2 FTOH few data are available, but many studies are underway. Results are expected by the end of 2002 (TRP, 2002).

6.4.1 Behaviour in humans

PFOS

PFOS was shown to be distributed in humans to serum and liver, where it is not metabolised. The excretion from the body is slow and occurs via urine and faeces (OECD, 2002) PFOS has an estimated excretion half-life in humans of 8.67 years. This is high compared to adult rats (100 days) and Cynomolgus monkeys (200 days). PFOS is well absorbed orally.

PFOA

PFOA has an estimated half-life between 1 and 3.5 years in humans. PFOA is well absorbed following oral and inhalation exposure and to a lesser extent following dermal exposure. As was observed in other biota, PFOA does not partition to the body fat, but covalently binds to macromolecules. In liver, plasma and kidney PFOA is not metabolised in the human body.

Urine and faeces are the primary routes of excretion for PFOA; female rats possess an unidentified extra mechanism for the excretion of PFOA. Therefore this chemical is excreted much faster in female rats than in male rats. The difference between sexes has also been observed in dogs, but not in primates and humans (USEPA, 2002). For perfluorocarboxylic acids the length of the perfluoroalkyl chain is important for the excretion. Perfluorocarboxylic acids with longer chain length are less eliminated (Kudo et al, 2001).

6.4.2 Acute toxicity

PFOS

The available rodent toxicity data of PFOS have been summarised in table 6.12.

	Species	Result (mg/kg)
Oral	Rats	LD_{50} = 251
	Rats	1h LC_{50} = 5.2
Eye irritation	Rabbits	Mildly irritating
Skin irritation	Rabbits	Non-irritating

Table 6.12 Acute toxicity data of PFOS to rodents

PFOA

The available rodent toxicity data of PFOA have been summarised in table 6.13.

	Species	Result (mg/kg)
Oral	CD Rats	LD_{50} > 500 (male) LD_{50} 250-500 (female)
	Wistar rats	LD_{50} < 1000 (female)
Inhalation	Rats	1h NOEC > 18,6 mg/L
Dermal	Rabbits	LD_{50} > 2000 mg/kg
Eye irritation	Rabbits	Irritating
Skin irritation	Rabbits	Irreversible tissue damage
	Rabbits	Non-irritating

Table 6.13 Acute toxicity of PFOA to rodents

6.4.3 Chronic toxicity

PFOS

In repeat-dose oral toxicity studies with PFOS using rats and primates the exposure resulted in hepatotoxicity and mortality. At an exposure level of from 2mg/kg/day an above observed effects in rats are increases in liver enzymes, hepatic vacuolisation and hepatocellular hypertrophy, gastrointestinal effects, haematological abnormalities, weight loss, convulsions and death. These effects were confirmed by a 2-year bioassay with rats. The lowest observed adverse effect level (LOAEL) in female rats was 5 ppm; the associated no observed adverse effect level (NOAEL) was 2 ppm. In male rats the LOAEL was 0.5 ppm; no NOAEL could be determined. In a developmental effect study the NOAEL and the LOAEL for the second generation of rats were determined to be 0.1 mg/kg/day and 0.4 mg/kg/day, respectively (OECD, 2002).

In repeat-dose oral toxicity studies with PFOS using Rhesus monkeys the effects observed included anorexia, emesis, diarrhoea, hypoactivity, prostration, convulsions, atrophy of the salivary glands and the pancreas, marked decreases in serum cholesterol, and lipid depletion in the adrenals. These effects were observed at levels from 1.5 mg/kg/day and above. No survival was reported after three weeks treatment with 10 mg/kg/day and after seven weeks with 4.5 mg/kg/day. In a six-month study no effects were observed at doses of 0.15 or 0.03 mg/kg/day (OECD, 2002).

In mutagenicity with S. typhimurium, E. coli, human lymphocytes, rat hepatocytes and mouse micronucleus, PFOS was found to be non mutagenic (OECD, 2002).
In a 2-year carcinogenicity assay with Sprague-Dawley rats significant increase in the incidence of hepatocellular adenomas was observed at the highest dose of 20 ppm of PFOS (OECD, 2002).

PFOA

In various studies with S. Typhimurium, E. Coli and human lymphocytes PFOA was found to be non-mutagenic induce mutations. PFOA was negative in an essay with mouse embryo fibroblasts and in an in vivo mouse micronucleus assay.
PFOA did induce chromosomal aberrations and polyploidy in CHO cells (USEPA, 2002).

Sub-chronic studies in rats and mice showed that the liver is the primary target organ. Observed effects are increased liver and kidney weight, hepatocellular hypertrophy, at 1000 ppm for female rats (76.5 mg/kg/day) and 100 ppm for male rats (5 mg/kg/day).

Studies with rhesus monkeys resulted in death, lipid depletion in the adrenals, hypoplasia of the bone marrow, and moderate atrophy of the lymphoid follicles in the spleen and lymph nodes at 30 mg/kg/day or higher (USEPA, 2002).

Rats fed with 300 ppm PFOA showed increased liver and kidney weight, haematological effects and liver lesions in males and females. In addition, increases in testicular masses (males at 300 ppm) and ovarian tubular hyperplasia (females at 30 ppm) were observed (USEPA, 2002).

Carcinogenity studies with rats showed that PFOA is weakly carcinogenic, inducing Leydig cell adenomas in the males and mammary fibroadenomas in the females following 2-year exposure to 300 ppm. At that level PFOA has also been reported to be carcinogenic to the liver and pancreas of male CD Rats (USEPA, 2002).

Telomers (based on TRP, 2002)
For a mixture of 6:2 FTOH, 8:2 FTOH, 10:2 FTOH and 12:2 FTOH three NOELs have been determined. The repeated dose and the reproductive toxicity NOEL was 25 mg/kg/day. No developmental toxicity was observed at 200 mg/kg/day.
Furthermore these substances reacted negative in the AMES, Chrom Ab geno-toxicity tests.

6.5 Conclusions and recommendations

Various data were available to determine the toxicity of PFOS and PFOA. The reliability of many of these tests must be considered as limited, because nominal concentrations were used. Due to the special, high sorptive behaviour, the actual concentration may have been significantly reduced.

PFOS is moderately acute toxic to freshwater fish and invertebrates. Toxicity to algae is relatively low. The chronic toxicity of PFOS to freshwater fish and invertebrates is moderate. PFOS is moderately toxic to marine invertebrates (acute and chronic) and algae (acute). The derived $iMPC_{freshwater}$ is 6 •g/L. The suggested iMPC for marine water is 1.1 •g/L. PFOS concentrations were shown to exceed the iMPC, in point source receiving fresh water. In other freshwaters, the iMPC was approached.
The acute toxicity of PFOA to freshwater invertebrates and algae is moderate, whereas the toxicity to freshwater fish is relatively low. An $iMPC_{freshwater}$ for PFOA of 3.8 •g/L has been derived. This iMPC can be exceeded due to point sources.
PFOSGE has a high acute toxicity to freshwater invertebrates and moderate toxicity to fish. An $iMPC_{freshwater}$ has been derived of 0.29 •g/L. This iMPC could not be compared with sampling data. N-EtFOSA exhibits moderate acute toxicity to invertebrates and low toxicity to fish. PFDS is moderately acute toxic to fish and invertebrates.

No effects have been observed for 8:2 FTOH. No conclusions regarding the toxicity of this substance can be drawn, since nominal concentration have been used

Concerning humans, both PFOS and PFOA have long half-lives (8.67 and 1-3.5 years, respectively) in the human body. Both chemicals are distributed to liver, plasma and kidney. To rodents PFOS and PFOA exhibit low acute toxicity, but they are eye irritating.

In chronic feeding tests with rodents and primates the primary target was the liver for PFOS and PFOA. PFOA was found to be weakly carcinogenic. Mutagenicity testing of

PFOS did not show any mutagenic effects. PFOA did not show mutagenic effects in most mutagenicity tests, but did induce chromosomal aberrations and polyploidy in CHO cells.

In a developmental effect study with PFOS the NOAEL and the LOAEL for the second generation of rodents were determined to be 0.1 mg/kg/day and 0.4 mg/kg/day, respectively.

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7 Policy and governmental awareness

7.1 National Environmental policy

7.1.1 Netherlands

In the National Environmental Policy Plan (NMP, 1989) and the more recently published National Environmental Policy Plan-3 (NMP-3, 1997) the general environmental policy of the Netherlands is described.
By the year 2010 the environmental targets and target values must have been reached. Concerning the reduction of the risks caused by high concentrations of chemicals, specific policy targets have been set in the National Environmental Policy Plan of 1989. The targets imply the aim to not exceed the Maximum Permissible Concentrations (MPCs) and the Negligible Concentrations (NCs) in 2010, by means of prevention and reconstruction of production processes. These values are guidelines but not legally binding. When the environmental quality standards are set, other aspects, such as political and technical feasability, are also taken into account. Target values are either set at the NC or at the background value.
In the report on integral standardisation on substances (INS, 1997) environmental quality standards have been derived. For PFS no specific quality standards, MPCs or NCs have been set.
The current water policy is reflected in the Fourth Note on Watermanagement (1997). In this note the targets and headlines of the policy for the national water management are given.

7.1.2 Other country specific policy/ governmental awareness

United States of America

Significant new use rule

The United States of America Environmental Protection Agency (USEPA) has initiated a significant new use rule (SNUR) for perfluoroalkyl sulfonates. It concerns 13 chemicals[3], including polymers that are derived from perfluorooctanesulfonic acid and its higher and lower homologues. The rule requires manufacturers and importers to notify the new use of these chemicals to USEPA, giving the USEPA the opportunity to evaluate the intended new use and associates activities (USEPA, 2002).

Hazard Assessment PFOA

The USEPA has performed a hazard assessment on PFOA. The corrected draft version has been released on April 15, 2002 and is under discussion.

Canada

The Canadian government is performing a environmental screening assessment on perfluoroalkyl substances for possible priority chemicals. This assessment is to be completed in Autumn 2002 (Windle et al, 2002).

United Kingdom

[3] The CAS-numbers of the concerning chemicals are: 2250-98-8, 30381-98-7, 57589-85-2, 61660-12-6, 67969-69-1, 68608-14-0, 70776-36-2, 127133-66-8, 148240-78-2, 14868-79-1, 178535-22-3, P-94-2205, P-96-1645 306974-63-0

The National Centre for Ecotoxicology & Hazardous Substances of the United Kingdom has reviewed the occurrence and hazards of perfluoroalkylated substances in the UK in 2001. It has been initiated as a response to the decision of 3M to phase out the perfluorooctanyl chemistry. This study takes a broader perspective and tries to incorporate the telomers as well (NCEHS, 2001).

Denmark
The Danish EPA has performed a survey of perfluorooctyl substances in consumer products. In three out of 21 purchased consumer products fluorinated chemicals were detected (PFDS, FOSA and n-EtFOSE) (NERI, 2002).

7.2 International policy/ awareness

7.2.1 OECD
The Organisation for Economic Co-operation and Development (OECD) is carrying out a hazard assessment on PFOS and its salts. The draft version of May, 13, 2002 is being discussed in the OECD task force of existing chemicals. Once the information is available, this will be followed by a risk assessment. Accordingly decisions will be taken on the need for international risk management (NCEHS, 2001).

7.2.2 OSPAR
The OSPAR Convention for the Protection of the Marine Environment of the Northeast Atlantic has performed a selection process for possible bioaccumulative, persistent and ecotoxic substances. Candidates were selected from a Danish QSAR database (Tyle et al, 2001, Tyle et al, 2002). About 60 perfluorinated chemicals were selected, out of a total of 92 possible substances (NCEHS, 2001).

7.3 Actions of industry

7.3.1 3M studies
3M has performed many studies on toxicology, pharmaco-kinetics and environmental fate and effects of perfluorinated chemicals. They have submitted the results of these studies with the USEPA, and discussed the results with them (3M, 2000). These data are available from USEPA (USEPA, 2001).

7.3.2 Telomer Research Program
The united perfluorinated telomer manufacturers (Asahi Glass, Atofina, Clariant, Daikin and Dupont) have set up a research program on the principal raw material common amongst the TRP members: 8:2 FTOH. The program focuses on three parallel work streams: toxicology, pharmaco-kinetics and environmental date and effect studies. Publication in the open literature of study results is encouraged. It is anticipated that the current research plan will take two more years to complete (TRP, 2002).

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List of Annexes

Annex 1 List of abbreviations

6:2 FTA	1H,1H,2H,2H perfluorooctyl acrylate
6:2 FTMA	1H,1H,2H,2H perfluorooctyl methacrylate
6:2 FTOH	1H,1H,2H,2H perfluorooctanol
8:2 FTA	1H,1H,2H,2H perfluorodecyl acrylate
8:2 FTMA	1H,1H,2H,2H perfluorodecyl methacrylate
8:2 FTOH	1H,1H,2H,2H perfluorodecanol
10:2 FTOH	1H,1H,2H,2H perfluorododecanol
12:2 FTOH	1H,1H,2H,2H perfluorotetradecanol
AFFF	Aqueous Film Forming Foam
APME	Association of Plastic Manufacturers Europe
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BMF	Biomagnification Factor
BOD	Biological Oxygen Demand
COD	Chemical Oxygen Demand
DDA	Dodecyldemethylammonium salt
DRI	Data Reliability Indicator
EC_{50}	Concentration that causes an effect for 50% of the tested organisms
ECD	Electron Capture Detection
ECF	Electrochemical Fluorination
EL_{50}	Level that causes an effect for 50% of the tested organisms
EP	Equilibrium partition
EQC	Equilibrium Criterion (Model)
FD	Fluorescence detection
FOSA	Perfluorooctane sulfonamid
GC	Gas Chromatography
HPLC	High-Pressure Liquid Chromatography
IC_{50}	Concentration that inhibits 50% of the tested organisms
iMPC	Indicative Maximal Permissible Concentration
LC	Liquid Chromatography
LC_{50}	Concentration that is lethal for 50% of the tested organisms
LL_{50}	Level that is lethal for 50% of the tested organisms
LOAEL	Lowest Observed Adverse Effect Level
LOD	Limit of Detection
LOQ	Limit of Quantification
MPC	Maximal Permissible Concentration
MS	Mass Spectrometry
n-EtFOSA	n-Ethyl perfluorooctane sulfonamid
n-EtFOSE	n-Ethyl perfluorooctane sulfonamidoethanol
n-EtFOSEA	n-Ethyl perfluorooctane sulfonamidethyl acrylate
n-EtFOSEMA	n-Ethyl perfluorooctane sulfonamidethyl methacrylate
n-MeFOSE	n-Methyl perfluorooctane sulfonamidethanol
n-MeFOSEA	n-Methyl perfluorooctane sulfonamidethyl acrylate
NMR	Nuclear Magnetic Resonance
NOAEL	No observed Adverse Effect Concentration

NOEC	No observed Effect Concentration
NOEL	No observed Effect Level
OECD	Organisation for Economic Co-operation and Development
PFBS	Perfluorobutyl sulfonate
PFDS	Perfluorodecyl sulfonate
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxS	Perfiluorohehexyl sulfonate
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctyl sulfonate
PFOSGE	n-perfluorooctylsulfonyl-N-ethylglycinate
PFS	Perfluorinated Surfactants
POSF	Perfluorooctane sulfonyl fluoride
POTW	Publicly owned treatment plant
ppb	Parts per billion
ppm	Parts per million
PTFE	Polytetrafluorethylene
QSAR	Quantitative Structure-Activity Relationship
RIKZ	Rijksinstituut voor Kust en Zee (Insititute for Coastal and Marine Management)
SNUR	Significant New Use Rule
TFE	Tetrafluoroethylene
TGD	Technical Guidance Document
TRP	Telomer Research Project
USEPA	United States Environmental Protection Agency
VNTF	Vereniging van Nederlandse Tapijt Fabrikanten (Association of Netherlands Carpet Manufacturers)
VTN	Vereniging Textielindustrie Nederland (Dutch Association for the Textile Industry)
WWTP	Wastewater Treatment Plant

Annex 2 Data Reliability Indicator

Data Reliability Indicator

In the data that were gathered for this study large discrepancies were found in values for comparable properties. Although different test methods mostly result in different outcome, well-conducted experiments should give values in the similar range. Several researchers have tried to develop indicators for data quality. For the present study two important publications on this subject have been used: Kollig (1988) and Klimisch et al (1997). Both researchers describe indicators for evaluating data reliability.

Kollig (1998) divides the indicator in four categories:

1. Analytical information
2. Experimental information
3. Statistical information
4. Corroborative information

Ad 1. Was the analytical method appropriate and suitable for the particular compound? If no standard method has been used, is the method sufficiently described?
Ad 2. Are all experimental parameters (temperature, pH, purity, etc.) well stated? Is the chemical identified by testing?
Ad 3. Is the uncertainty and the reproducibility of the test mentioned?
Ad 4. Are the data in accordance with the results of another independently conducted study?

Each category contains subcriteria that are developed for various properties that make it possible to estimate the reliability of the measurement within one category. The Data Reliability Indicator (DRI) consists of the relative reliability for all four categories.

Klimisch et al (1997) use four reliability scores for experimental data-generating studies:

1. Reliable without restrictions
2. Reliable with restrictions
3. Not reliable
4. Not assignable

Ad 1. The tests are performed according to internationally accepted test guidelines and preferably in compliance with Good Laboratory Practice (GLP).
Ad 2. The tests are not entirely performed according to internationally accepted test guidelines. Nevertheless the conditions are acceptable. This category also includes investigations that have no official testing guideline, but that are scientifically acceptable.
Ad 3. The test designs that are assigned to this category can have interference between the test substance and the measuring system or the test system is not relevant in relation to the exposure or the test method is not acceptable.
Ad 4. No reliability can be assigned if insufficient experimental details are given.
For various tests subcriteria are supplied for the evaluation of tests that were executed not according to internationally accepted test guidelines to be assigned 'reliable with restrictions'. Nevertheless, data in category 3 or 4 can very well be used as corroborative information, or as a 'first estimation' if no other data are available.

Both methods can be useful in the assessment of reliability. The Kollig method does not supply a final judgement of reliability; the Klimisch method does not give a detailed set of criteria. All data that are supplied by the 3M company have been evaluated with the Klimisch ranking system. Furthermore, most of the data-generating experiments in the present study have been performed (partially) according to ISO, OECD or EPA testing. Therefore it will more practical to use the Klimisch ranking system.

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Annex 3 Production processes

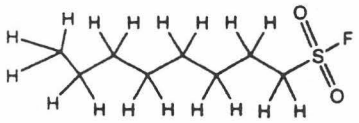
Introduction

Perfluorinated surfactants can be produced by two routes of synthesis; fluorination of organic compounds, in which hydrogen atoms of non-fluorinated or partially fluorinated organic compounds are substituted by fluorine atoms (Moldavsky et al, 1999), or reactions with perfluorinated compounds to form PFS. Two important routes of production are used commercially: 1) electrochemical fluorination and 2) telomerisation. Also methods of fluorination using high-valence metal fluorides (CoF_3, MnF_3, AgF_2) or elemental fluorine (F_2) are known (Field, 1994, Moilliet, 2001), but these techniques are not important for the commercial synthesis of surfactants. In the following paragraphs the first two routes of synthesis of perfluorinated surfactants will be discussed.

Electrochemical fluorination

The electrochemical fluorination is being used by the 3M company, and will be terminated for the largest part by 2003 (3M, 2000a). In this reaction an organic compound is introduced in liquid anhydrous hydrogen fluorine (aHF) at nickel anodes. An electric current is led over these electrodes, resulting in the substitution of the hydrogen atoms of the organic compound by fluorine atoms. This method was developed by Simons et al in 1944 (3M, 2002). 3M bought the patent immediately, but did not have any commercial application until 1956 (Riecher, 2000). Since then it has been used as a commercial process by 3M for more than 40 years (Noel et al, 1996).

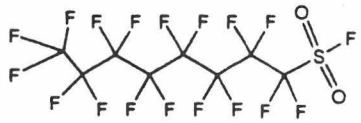
The overall reaction is the following:



$C_8H_{17}SO_2F + 17\ HF$

1-Octanesulfonyl fluoride

$+ 17\ HF \rightarrow$



$\rightarrow C_8F_{17}SO_2F + 17\ H_2$

Perfluorooctanesulfonyl fluoride (POSF)

and exists of two subreactions, at the anode and cathode (Alsmeyer et al, 1994):

anode: $C_mH_nX + n\text{HF} \rightarrow C_mF_nX + 2n\text{H}^+ + 2n\text{e}^-$

cathode: $2n\text{H}^+ + 2n\text{e}^- \rightarrow n\text{H}_2$

POSF is further reacted with methyl or ethyl amine, resulting in N-ethyl (and methyl) perfluorooctanesulfonamide (N-EtFOSA), and subsequently with ethylene carbonate to form either N-methyl or N-ethylperfluorooctanesulfonamidoethanol (N-EtFOSE). N-EtFOSE and N-MeFOSE are the principal building blocks of 3M's product lines (3M, 1999).

Various sources provide estimations of the yield of the fluorination of 1-octanesulfonyl fluoride (3M, 1999, 3M, 2000b, 3M, 2001).

35-40%	n-POSF
20-25%	Perfluorinated alkanes and ethers
18-20%	Branched non-C8 perfluorinated sulfonyl fluorides
10-15%	Tars (high molecular weight fluorochemical byproducts) and molecular hydrogen
7%	Linear non C8-perfluorinated sulfonates

Table III.1 Impurities in POSF production

These percentages may vary from plant to plant, due to differences in raw materials, equipment and process conditions. The tars and non functional molecules are easily removed from the reaction mixture. The final product will contain approximately 70% n-POSF and 30% branched impurities (3M, 2000b).

The impurities can be due to impurities in the reactant or rearrangement during fluorination. Although n-octanesulfonyl fluoride is used, there are always traces of other C8 compounds, leading to non-linear POSF. However, their presence does not affect the application properties (Moldavsky & Furin, 1998). Similar impurities can be expected in PFOA production. PFOA is produced via the electrochemical fluorination of octanoyl chloride to perfluorooctanyol fluoride. This is hydrolysed to PFOA.

Other impurities can be partially fluorinated. This is due to the production process itself: '[..] *Simons processes* [is] *a step by step fluorination process which leads to the formation of all possible partially fluorinated compounds [..]' (Sartori & Ignatiev, 1998).*

According to several sources (Moldavsky & Furin, 1998; Moldavsky et al, 1999; 3M, 1999, 3M, 2001) also non-C8 compounds can be found: *'[..] fragmentation and*

rearrangement of the carbon skeleton can also occur and significant amounts of cleaved, branched and cyclic structures may be formed.' (3M, 1999). Fragmentation of the carbon framework is to be expected, because the energy of the C-F bond formation exceeds that of the C-C bond (Moldavsky et al, 1999). With electrochemical fluorination perfluorinated compounds with even and odd numbers of perfluorocarbon atoms are generated (Kauck & Diesslin, 1951 cited in Moody & Field, 2000; Kissa, 2001).

The commercially available POSF contains more than 90% of C8-molecules, of which approximately 25% is branched. The perfluorinated C6 compounds constitute 5-10% of the POSF product and the remainder is C7 (2-3%) and C5 (3M, 2001). The distribution of chain length is assumed to be comparable for the fluorination of octanoic acid to form PFOA.

Telomerisation

The second important route of synthesis of PFS is telomerisation. This process is used by AtoFina, DuPont, Clariant, Daikan and Asahi Glass (Wakselman & Lantz, 1994; AtoFina, 2001). Telomerisation is a process in which '[..] *a polymeric product* [is] *formed from a monomer and an initiator, R, obtained by a chain-transfer reaction between a radical from a catalyst and some other compound, called a telogen.'* (Kirk & Othmer, 1954). In the first stage of this production process perfluoroalkyl iodides are synthesised. In the second the iodide is substituted by a functional group, depending on the application.

The first stage of the process, the manufacturing process for the perfluoroalkyl iodide, involves two steps:

1) $5\ C_2F_4 + IF_5$ (from $I_2 + F_2$) $+ 2I_2 \xrightarrow{catalyst} 5\ C_2F_5I$

2) $C_2F_5I + nC_2F_4 \rightarrow C_2F_5(C_2F_4)_nI$

The second step uses a radical-initiated mechanism. This can be initiated using heat, UV light or radical sources (Wakselman & Lantz, 1994). This manufacturing process is developed by Haszeldine in 1949 and adapted by the DuPont company in the 1960s (Rao & Baker, 1994).

The price of compounds produced via this production route is high. The main reasons are the properties of the starting materials. The I_2 and IF_5 are highly aggressive and the tetrafluorethylene is expensive and potentially explosive (Wakselman & Lantz, 1994).

In the second stage of production the iodide has to be substituted with a functional group. Only two important commercial products can be produced *directly* from C_nF_mI, being perfluorocarboxylic acid (using oleum as reactant) and perfluoroalkanesulfonyl chloride (using SO_2/Zn and Cl_2). Indirectly products can be produced by ethylenation, followed by substitution of the iodide by a functional group of choice, thus forming $R_fC_2H_4X$ (Wakselman & Lantz, 1994), where R_f represents a perfluorinated alkyl group. The compounds that are produced via this indirect route are the most important intermediates for perfluorinated surfactant production, with 1*H*,1*H*,2*H*,2*H*-perfluorodecanol (8:2 FTOH, see figure III.2) as primary building block.

During the telomerisation also C4 and C6 iodide can be formed by the radical reaction. Two other important possible by-products can be formed with this production process, due to the following reactions (Rao & Baker, 1994):

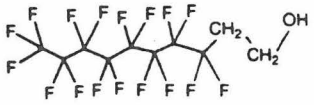


Figure III.2. 8:2 FTOH

1). $R_fI + IF_5 \rightarrow R_fF + (I_2F_4)$

2) $2\ R_fI \rightarrow R_fR_f$

All undesired products are removed by distillation. This is a simple process. Because of the radical mechanism, only linear perfluoro-*n*-alkyl compounds are to be expected.

Comparison of production processes

The most important difference between the two major production processes of PFS, is the final product. Electrochemical fluorination can produce all types of PFS and will be largely dependent on the starting organic material that is used, and its purity. It was used by 3M to produce POSF and PFOA. Almost all products that are synthesised using telomerisation have $R_fC_2H_4X$ as an intermediate, in which X represents any functional group.

When electrochemical fluorination and telomerisation are compared, also the purity of the final products is an important difference. The products from the telomerisation process are more pure than the products formed via electrochemical fluorination. The telomerisation process gives fewer by-products and furthermore it is easier to separate those from the desired product, so that relatively pure products are obtained. The perfluorinated products from the electrochemical process yield both even and odd numbered perfluorinated carbons, in contrast to the perfluorochemicals that are

synthesised via telomerisation, which have only even numbers of perfluorinated carbon atoms (Kissa, 2001).

Last, there is a price difference between the two processes. Telomers are exceptionally expensive products (Wakselman & Lantz, 1994), whereas the electrochemical fluorination process is relatively cheap (Hudlicky & Pavlath, 1995; cited in Moody & Field, 2000). Exact figures are not available.

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Annex 4 Mechanism of AFFF

AFFF is used by several types of fire fighters, including fire departments at airports, military, chemical plants and off-shore drilling platforms (3M, 1999a, Moody & Field, 2000). The products are also called *light water*, because they form a film on the burning fluid.

The fire fighting mechanism of foam is based on four principles (Luttmer, 1998):

1. The capability to seal the surface and isolate it from contact with atmospheric oxygen,
2. Thermal stability,
3. Low density,
4. Cooling by the water that percolates through the foam.

The first two principles are partially based on the properties of fluorochemicals. As stated earlier surfactants form micelles in water. Perfluorinated surfactants form lamellar micelles, thus perfectly covering the burning fluid with the foam (Pabon & Corpart, 1999; Moody & Field, 2000).

The foam provides better grip to the material in flames, producing a continuous cover (Figueredo et al, 1999). The combination of hydrocarbon and perfluorinated surfactants is responsible for the covering. '*The films formed by fluorocarbon and hydrocarbon solutions consist of two mixed monolayers of surfactants where the air-aqueous phase monolayer is dominated by the fluorocarbon surfactant and the aqueous-hydrocarbon phase is dominated by the hydrocarbon surfactant* (see figure IV.1) (Moody & Field, 2000).'

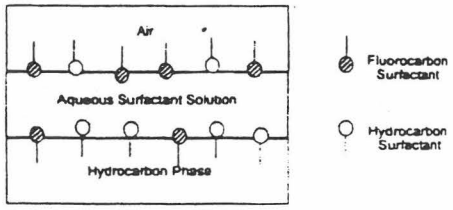

Figure IV.1. The mechanism of fire fighting foams (Moody & Field, 2000)

The film that is formed is less permeable for heptane vapours than the films formed by hydrocarbons surfactants, thus preventing re-ignition of the fuel (Pabon & Corpart, 1999, Moody & Field, 2000).

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Annex 5 Non-reliable toxicity tests

Annex 6 Derivation of iMPC

PFOS

For PFOS two chronic NOECs are available, covering the trophic levels that showed the lowest acute $L(E)C_{50}$. Therefore, following the TGD method (ECB, 1996), an assessment factor of 50 is applied to the lowest NOEC, being 0.30 mg/L (Fathead minnow).

Therefore, the $iMPC_{freshwater}$ is 6 •g/L.

For the marine environment only one long-term NOEC is available. This NOEC is not for fish or daphnia (as is demanded in the TGD) but for Mysid shrimp. Therefore the marine iMPC has to be derived from the acute data. Although the LC_{50} for algae does not give a value, this test result can be used, because it is larger than the lowest marine $L(E)C_{50}$ for PFOS. The $L(E)C_{50}$ for invertebrates is not for Daphnia (as is demanded in the TGD). Therefore this value is also questionable. If however the suggested assessment factor of 1000 would be used, the $iMPC_{marine}$ would be 1.1 •g/L. This value is of the same order of magnitude as the derived freshwater iMPC.

PFOA

For PFOA no reliable chronic NOECs for fish or daphnia are available. Therefore the iMPC has to be derived from the reported $L(E)C_{50}$. The lowest acute $L(E)C_{50}$ is 3.8 mg/L for algae. Applying an assessment factor of 1000, this results in an iMPC of 3.8•g/L.

No marine data are available to derive an iMPC for marine water.

8:2 FTOH

For 8:2 FTOH insufficient data area available to derive an iMPC.

PFOSGE

For PFOSGE sufficient data are available to derive the iMPC, following the TGD method (ECB, 1996). Three acute $L(E)C_{50}$s are available; the lowest is 0.29 mg/L for Daphnia. Applying an assessment factor of 1000, the freshwater iMPC = 0.29 •g/L.

It has to be noted that the lowest observed $L(E)C_{50}$ was extrapolated from an impure test substance, using nominal concentrations. Therefore this iMPC has to be treated with some caution. If less than three $L(E)C_{50}$ are available the modified USEPA has to be used. Application of this method would result in the same iMPC.

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HOGANJH

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Kennedy
Charts from
5/7/91 results.

C-8, AMMONIUM PERFLUOROOCTANOATE

CURRENT AEL = 0.01 MG/M^3 (0.56 PPB) - SKIN BASED ON:

MODERATE ACUTE TOXICITY

DERMAL RESPONSE SEEN AT 1,500 MG/KG

SYSTEMIC RESPONSE FROM DERMAL DOSES OF 20 MG/KG TO RATS (G.L.K TAP 81:348:85)

HUMAN BLOOD LEVELS DECLINE SLOWLY - t 1/2 IN EXCESS OF 1 YEAR

INHALATION NO-OBSERVED EFFECT LEVEL IS 1 MG/M^3

LIVER IS TARGET ORGAN IN RATS - DERMAL AND INHALATION

LARGER SAFETY FACTOR THAN USUAL SINCE C-8 PERSISTS IN HUMAN BLOOD

2 YEAR FEEDING STUDY IN RATS - 30 AND 300 PPM

LIVER DAMAGE, BOTH LEVELS

TESTICULAR TUMORS - 300 PPM

HORMONAL MEDIATION - NOEL DETERMINED

QUANTITATIVE ASSESSMENT

FROM RAT INHALATION - NOEL - 1 MG/M^3
(DOSE TO RAT = 0.005 MG/DAY)

FROM RAT ORAL - LOEL = 30 PPM
(DOSE TO RAT = 1 MG/DAY)

LO CANCER EL = 300 PPM
(DOSE TO RAT = 10 MG/DAY)

AT CURRENT AEL - HUMAN DOSE IS 0.1 MG/DAY

RECOMMEND AEL REMAIN AT 0.01 MG/M^3
WITH SKIN NOTATION

FOR COMMUNITY GUIDELINE

1) 8 VS. 24 HRS: MATERIAL CLEARS SLOWLY FROM HUMAN BLOOD

2) SENSITIVE SUBPOPULATION: TO LIVER TOXINS; DIFFICULT TO ESTIMATE

APPLY CHRONIC, SYSTEMIC TOXICITY CONCERNS

RECOMMEND CEG OF 0.0003 MG/M^3 = 0.3 μG/M^3

AMMONIUM PERFLUOROOCTANOATE (C-8)

EXISTING LIMITS

TLV● = 0.1 MG/M^3

AEL = 0.01 MG/M^3

CEG = 0.0003 MG/M^3 OR 0.3 µG/M^3

EXPOSED DAILY DOSE AT LIMITS - MAN

TLV● = 1 MG/DAY (0.014 MG/KG)

AEL = 0.1 MG/DAY (0.0014 MG/KG)

CEG = 6 µG (0.00009 MG/KG)

RJ7005428

EID078783

AMMONIUM PERFLUOROOCTANOATE

WATER GUIDELINE

DAILY EXPOSED DOSE FOR MAN IS APPROXIMATELY 6 µG/DAY

DRINKING WATER/INHALATION EXPOSURE % = 20/80

THEORETICAL AIR = 4.8 µG WATER = 1.2 µG

DAILY WATER INTAKE = 2 L

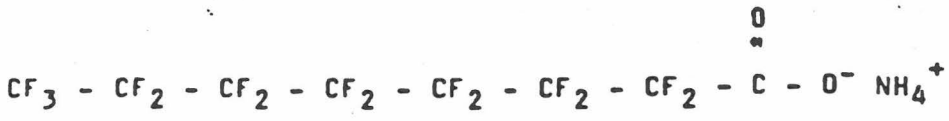
1.2 µG/2 L = 0.6 µG/L = 0.6 PPB

RECOMMENDATION

WATER GUIDELINE = 1 PPB

AMMONIUM PERFLUOROOCTANOATE

APFO, C-8



WHITE POWDER

1 PPM = 17.6 mg/m^3

1 mg/m^3 = 0.057 PPM

EXPOSURE STANDARDS

TLV 0.1 mg/m^3 - SKIN

AEL 0.01 mg/m^3 - SKIN = 0.56 ppb = 10 $\mu g/m^3$

CEG = 0.3 $\mu g/m^3$

RJZ005430

EID078785

ACUTE TOXICITY

- MODERATE ORAL TOXICITY

  LD50 470 MG/KG RATS

  457 MG/KG MICE

  178-217 MG/KG GUINEA PIGS

  > 200, < 450 MG/KG DOGS

RAT

- EFFECT OF AGE

  LD50 NEWBORN 243, 258 MG/KG (M/F)

  21 DAY OLD 573, 580

  8-WEEK OLD 470, 453

  1 YR OLD 336, 343

- EFFECT OF SURGICAL INTERVENTION

  LD50 400-491 mg/kg

  INTACT

  CASTRATED

  OVARIECTOMIZED RATS

- EFFECT OF LIVER STIMULATION

  LD50 WITH PhB 478 mg/kg (vs. 470)

  WITH PROADIFEN 452 mg/kg

ACUTE TOXICITY

SLIGHT DERMAL TOXICITY

LD50 RABBIT = 4,278 MG/KG

LD50 RATS = 6,959 MG/KG

SLIGHT TO MODERATE SKIN IRRITANT - RABBIT

MODERATE EYE IRRITANT - RABBIT (14/110)

ACUTE TOXICITY

MODERATELY TOXIC BY INHALATION

4 HR ALC - RATS = 800 MG/M^3

MODERATELY TOXIC BY INJECTION (IP)

LD50 - MICE = 192 MG/KG

REPEATED DOSE TOXICITY - ORAL

LIVER IS THE TARGET OF C-8 TOXICITY

- MICE
  - FED 2 WEEKS 10, 30, 100, 300, 1,000 3,000 OR 10,000 PPM
  - DEATHS 1,000 PPM OR GREATER
  - LIVER WEIGHT INCREASES DOSE - DEPENDENT TO 10 PPM
  - DOSED 3 WEEKS 0.1, 1, 10 MG/KG
  - DEATHS 10 MG/KG
  - LIVER WEIGHT INCREASES, 0.1, 1 MG/KG

- RATS
  - FED 13 WKS, MALES AND FEMALES, 10, 30, 100, 300, 1,000 PPM
  - BODY WEIGHT DEPRESSION - 300 + 1,000 MALES ONLY
  - LIVER SIZE INCREASE - 300 + 1,000 PPM, MALES ONLY
  - LIVER PATHOLOGY, MALES ONLY

- MONKEYS
  - DOSED 13 WKS, 3, 10, 30, 100 MG/KG
  - DEATH - 100 MG/KG
  - BODY WEIGHT LOSSES - 30 MG/KG
  - LIVER DAMAGE - 30 MG/KG, 10 MG/KG (MARGINAL)
  - 3 MG/KG, NOAEL
  - 10-30 MG/KG - HEMATOLOGIC CHANGES (MARGINAL)

REPEATED DOSE TOXICITY - DERMAL

RATS, MALES, 6 HR/DAY, 2 WKS - 0, 20, 200, 2,000 MG/KG
(+ 84 DAY RECOVERY)

WEIGHT LOSS - 200 - 2,000 MG/KG
SKIN IRRITATON - 2,000 MG/KG
LIVER DAMAGE - ALL GROUPS
LIVER WEIGHT DOSE-DEPENDENT
LIVER NECROSIS - 2,000 MG/KG

BLOOD ORGANOFLUORIDE - ELEVATED, DOSE-DEPENDENT
DECREASE DURING RECOVERY PERIOD - 84 DAYS

RJZ005435

EID078790

REPEATED DOSE TOXICITY - INHALATION

RATS (MALES), 6 HR/DAY, 5 DAY/WK X 2 WEEKS (84 DAYS RECOVERY) 0, 1, 8, 80 MG/M^3

MORTALITY - 80 MG/KG
BODY WEIGHT EFFECTS - 8 + 80 MG/M^3
LIVER DAMAGE - 80 MG/KG
LIVER WEIGHT INCREASE - 8 + 80 MG/M^3
BLOOD FLUORIDE - DOSE-RELATED INCREASE,
STILL SEEN 84 DAYS POST-EXPOSURE
ESTIMATED BLOOD 1/2 LIFE - 14-20 DAYS

DEVELOPMENTAL TOXICITY

- RATS, ORAL - 0, 25, 50, 75, 100, 150 MG/KG/DAY
  DAYS 6-15

  MATERNAL WEIGHT GAIN - DEPRESSED 150 MG/KG
  FETAL EFFECTS - NONE

- RATS, INHALATION, 6 HR/DAY, DAYS 6-15
  0, 0.1 - 1 - 10 - 25 MG/M^3

  MATERNAL EFFECTS - DEATH 25 MG/M^2
  BODY WEIGHT LOSS 10 MG/M^3

  FETAL EFFECTS - RESORPTION INCREASED 25 MG/M^3
  FETAL WEIGHT DECREASE 10 MG/M^3
  NO MALFORMED

  NOAEL 1 MG/M^3

(

no Adverse
Effect Level

GENETIC TOXICITY

- NON-ACTIVE IN AMES TEST

  SALMONELLA STRAINS TA 1535
  TA 1537
  TA 1538
  TA 100

- NOT ACTIVE IN SACCHAROMYCES CEREVISIAE

CARCINOGENICITY

RATS FED AT 0, 30, AND 300 PPM FOR 2 YEARS

- TESTICULAR TUMORS (LEYDIG CELL ADENOMAS)

0/50	0 PPM	(0%)
3/50	30 PPM	(6%)
7/50	300 PPM	(14%)

(0 PPM RANGE 0 - 12% $\bar{X}$ 6%)

- MAMMARY GLAND FIBROADENOMAS

10/50	0 PPM	(20%)
19/50	30 PPM	(38%)
21/50	300 PPM	(42%)

(0 PPM - $\bar{X}$ 37%)

- DECREASE IN BODY WEIGHT
- LIVER EFFECTS - BOTH LEVELS

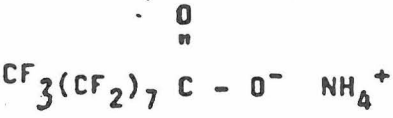
30 PPM MARGINAL EFFECT LEVEL LIVER

NOEL FOR TUMORIGENICITY

MECHANISM OF C-8 INDUCED TESTICULAR TUMORS

- TREATED MALE RATS WITH ORAL DOSES OF C-8
- FOUND DOSE-RELATED DECREASE IN ABSOLUTE AND RELATIVE SECONDARY SEX ORGAN WEIGHTS
- DOSE RELATED INCREASE IN SERUM ESTRADIOL LEVELS
- DOSE RELATED DECREASE IN SERUM TESTOSTERONE
  - APPEARS TO BE DIRECT EFFECT ON TESTES
  - HINDERED CONVERSION OF 17-OH PROGESTERONE TO ANDROSTENEDIONE (PRECURSOR OF TESTOSTERONE)
- GENETIC MECHANISM NOT INVOLVED

AMMONIUM PERFLUORONONANOATE (C-9)



WHITE SOLID

C-12 = 10 m

- MODERATE ACUTE ORAL TOXICITY - LD50 RAT = 407 MG/M^3
  C-10 ≈ 40
- MODERATE ACUTE INHALATION TOXICITY - ALC RAT = 590 MG/M^3
- REPEATED DOSE TOXICITY
  MOUSE LIVER WEIGHT INCREASE PRODUCED BY 1 PPM IN DIET (14 DAYS)
- LIVER TOXICITY
  LIVER WEIGHT INCREASE SEEN IN RATS EXPOSED TO 6 HOURS TO 67 MG/M^3
- GENETIC TOXICITY

  NO ACTIVE IN AMES SALMONELLA TEST

REASONING BEHIND CEG (COMMUNITY EXPOSURE GUIDELINE)

AS FOR THE AEL (0.01 MG/M^3)

- ADDITIONAL FACTORS
  1) 24 HR EXPOSURE IN COMMUNITY VS 8 HR AT WORK
  2) DIVERSE POPULATION IN COMMUNITY
  3) BIO-PERSISTENCE IN MAN

- RECOMMEND 0.0003 MG/M^3 (33 FOLD REDUCTION)

REASONING BEHIND AEL

- INHALATION NOAEL 1 MG/M^3

  EFFECTS AT 8 MG/M^3 MILD, REVERSIBLE

- LOW LEVEL (?) ACTIVITY IN TESTES (TUMORS)

  30 PPM IN DIET = 1.5 MG/KG/DAY

  100% ABSORPTION OF INHALED DOSE, 70 KG WORKER = 10.5 MG/M^3

  THIS IS 1) EFFECT LEVEL
  2) HUMAN CLEARANCE FROM BODY SLOW

  SAFETY (UNCERTAINTY) FACTOR SHOULD BE LARGE (1000)

- RECOMMEND 0.01 MG/M^3 (10.5 ÷ 1000)

RJZ005443

EID078798

REASONING BEHIND TLV

- LOW IN ACUTE TOXICITY
- INHALATION NOAEL 1 MG/M^3 (8 MG/M^3 EFFECT NOT PRONOUNCED)
- GENETIC TOXICITY/DEVELOPMENTAL TOXICITY - NONE
- CARCINOGENIC ACTIVITY - NOT DETERMINED
- MALE RAT RETAINS C-8/MAN RETAINS C-8
- DATA FROM 3M PRODUCER REPORTED NO ILL HEALTH EFFECTS - EXPOSURES FROM <0.03 TO 7.6 MG/M^3
- ESTABLISH 0.1 MG/M^3 AS TLV
- SKIN NOTATION: DEMONSTRATED LIVER EFFECTS FOLLOWING DERMAL TREATMENT

METABOLISM

- MAIN ISSUE IN MAN: PERSISTANCE IN BLOOD
  1/2 LIFE > 1 YR

- ANIMALS - SEX DIFFERENCE

  MALE RAT - EXCRETES 40% IN 120 HRS, BLOOD 1/2 LIFE 10-14 DAYS
  FEMALE RAT - EXCRETES 99% < 6 HRS, LITTLE TO BLOOD

  MALE HAMSTER - AS FEMALE RAT
  FEMALE HAMSTER - AS MALE RAT (60% IN 120 HRS)

  MALE/FEMALE RABBIT - EXCRETE RAPIDLY

  MALE/FEMALE MOUSE - EXCRETE ONLY 20% IN 120 HOURS

- DETAILS ON RAT

  FEMALE - ORAL, RAPID UPTAKE (PEAK 1-2 HOURS)
  TOTAL CLEARANCE 24 HOURS
  1 VS MULTIPLE DOSES = NO DIFFERENCE

  MALE - ORAL, RAPID UPTAKE, CLEARANCE SLOW (OVER 84 DAYS)

- MAN - ~~NO APPARENT DIFFERENCE~~ MALE VS FEMALE
  BLOOD HALF-LIFE MEASURED IN YEARS

RJZ005445

EID078800

C-9

THE TOXICITY OF C-9 APPEARS SIMILAR TO C-8

BASED ON ANALOGY TO C-8, AEL OF 0.01 MG/M^3 ESTABLISHED

From: ZIPFEL --WWPS Date and time 07/15/93 08:32:00
To: RITCHERL--ISCDCVM2

From: ZIPFEL --WWPS A1::ZIPFEL
Subj: C-8 Programs

From: NAME: Roger J. Zipfel
FUNC: PPD-SPD
TEL: 304-863-2567 <ZIPFEL AT A1 AT WWPS>
To: NAME: Robert L. Ritchey <RITCHERL AT ISCDCVM2>

Here is the program status list.

Roger.

Author: Roger J. Zipfel
Date: 17-Jun-1993
Posted-date: 15-Jul-1993

Global C-8 Team
Program Status - May 31, 1993

I. We will comply with all federal, state, and local regulations governing the use and disposal of C-8.

A. Complete Verification Investigation plan for the Washington Works Supernate pond Solid Waste Management Unit (SWMU).

Status - complete

B. Develop program options for the reduction of C-8 in the aquifer below the Washington Works site.

Responsibility - Terry VanDell, Roger Zipfel
Timing - 1993
Status - Current study ongoing with the Univ of Del on electrochemical decomposition of C-8. Phase I of study is complete. Phase II, site specific study of feaseability of the technology at the

Washington Works, has begun. The study is using actual C-8 ladden

soil overlying the site's affected aquifer.

C. Determine level of C-8 in the ground water below the Dordrecht site.

Responsibility - Charles Mulder
Timing - 3Q93
Status - Initial round of samples have been taken and analysed. Samples show low levels of C-8 to be present. Data and hydrogeological information now being analysed to determine if further efforts are required.

II. We will control C-8 exposure to personnel by being in full compliance with the AEL (0.01mg/M3) for employees and with the CEG (0.0003 mg/M3 in air, and 1PPB in drinking water) for the general public.

A. What is the corporate definition of a CEG?

Status - Complete - First and second drafts issued. Draft definition is adequate to meet program needs.

B. Determine analytical method to be used for routine C-8 in water samples. Determine where the routine C-8 in water samples are to be analyzed.

Timing - Complete - CH2MHill laboratory chosen to analyze routine C-8 in water samples.

C. Define C-8 levels in the Delaware River.

Responsibility - Fabiola Sepulveda
Timing - Complete
Status - Second set of samples taken which show levels at the New Jersey side to be 3ppb. No further test work is planned at this time.

D. Complete study of C-8 accumulation in aquatic animals.

Responsibility - Gerry Kennedy, Fabiola Sepulveda
Timing - 9/93
Status - Study request has been approved. Start of study was delayed until 6/93.

E. Determine need for an update of the Washington Works' Employees epidemiological study for the affects of C-8.

Complete - Agreement made that study was not required at this time. Relook at need for a study in 1993.

F. Audit C-8 using sites for compliance to the MSDS and Dupont's AEL.

Responsibility - Roger Zipfel and site leader
Timing - TBD
Status - MDF visit conducted in May,1992, Dordrecht and Mechlen in September, 1992. Will schedule Chambers Works in 1993.

G. Establish a No Effect Level for C-8 relative to liver functionality.

Status - Complete. The NOAEL was established to be 1 ppm. This level verifies the duPont AEL of .01mg/M3. Haskell will now work to set the TLV at the AEL level.

H. Eliminate high C-8 in water use by single family on private well on the western edge of the Washington Works.

Responsibility - Woody Ireland, Dave Ramsey,Walt Stewart
Status - Complete.

I. Institute routine C-8 in air monitoring at Shimizu

Responsibility - S. Amemiya, Roger Zipfel
Timing - 3Q92 with statistical analysis by 3Q93
Status - Sampling began in Feb, 1993. Will review data on visit later in 1993.

J. Implement use of liquid C-8 at Dordrecht

Responsibility - Rob Rasenberg
Timing - 1993
Status - Starting negotiations with suppliers for liquid C-8.

K. Implement use of liquid C-8 at Shimizu

Responsibility - Akito Abe
Timing - 3Q93
Status - Scope development in progress.

III. We will communicate C-8 information to employees as determined by site management and to the general public as determined by site management with the advice of corporate external affairs and legal.

A. Complete update of the MSDS sheets for TFE and FEP dispersions relative to C-8.

Status - Complete. MSDS sheets for TFE dispersions have been updated and issued.

B. Communicate status of Washington Works ground water learnings to local public organizations.

Responsibility - Dave Ramsey
Status - Complete

C. Develop communication package on C-8 for MDF.

Responsibility - Roger Zipfel
Status - Will utilize material to be presented to Dordrecht by Kennedy, and Zipfel on October 2,1992.

IV. We will continue to use C-8 for the manufacture of fluoropolymers/ fluoroelastomers. Where feaseable, we will seek to replace C-8 with other more enviromentaly safe Materials.

A. Develop a maintenance sampling plan for C-8 in the enviroment for Washington Works, Dordrecht Works and Shimizu.

Responsibility Roger Zipfel
Timing - Washington Works - Complete
Dordrecht Works - 1993
Shimizu - TBD
Status - Dordrecht is presently defining scope of C-8 contamination. No need for this effort defined for Shimizu.

B. Evaluate the use of Zonyl C-6 TBS

Responsibility - Roger Zipfel
Timing - TBD
Status - Chemicals has suggested that Polymers examine the use of Zonyl C-6 TBS (6,2 TBS) as a possible alternative. Haskell has

determined that the new material is less toxic. Bioaccumulation in rat tests is significantly less. Test work at Washington Lab has demonstrated the feaseability of the use of 6,2 TBS in FEP. A plant test is now being scheduled. Additional work is needed for dispersion type products.

V. We will have a continuous improvement effort to reduce C-8 emissions to the Enviroment.

A. Improve efficiency of the Fine Powder drier C-8 recovery unit.

Responsibility - Thu-van Dihn, Mike McClusky
Timing - continuing
Status - Operation of scrubber for 1992 fell to 78% utility, system C-8 recovery fell to 25% of total C-8 fed to the driers. Goal scrubber utility is greater than 90%, with total recovery goal of 85%. Recovered C-8 is being reprocessed by 3M and then reused on site.

B. Develop Scope to Reuse C-8 from PTFE supernate

Responsibility - Roger Zipfel
Timing - 8/93
Status - Have developed capabilty to remove solids from the waste supernate stream (this work done in conjunction with E. Mayer, ESD). Working on basic data development for the separation of C-8 from Triton tm in the remaining aqueous stream.

C. Reduce C-8 levels from the Washignton Works FEP plant by >95%.

Responsibility - Nick Bittner, Roger Zipfel
Timing - 10/93
Status - Concept definition is complete. Calgon Carbon test work demonstrates that C-8 will be adsorbed by activated carbon. Solids removal in effluent stream is required. Solids removal basic data has been completed. Project for adsorption of most of the Washington Works effluent C-8 is currently on hold pending results of test work with 6,2 TBS in FEP.

D. Determine capability of the Chambers Works Waste Water Treatment Plant to remove C-8.

Responsibility - Fabiola Sepulveda
Status - Complete
Conclusions - Data analysis showed that the capability for this facility to remove C-8 was very limited. Carbon adsorption of C-8 is inhibited by the overwhelming amounts of other organics present which compete for adsorption sites.

E. Complete definition of C-8 contamination of the Letart and Dry Run land fills.

Responsibility - Dan Weber, Terry VanDell, Walt Stewart
Timing - First phase is complete
Status - New monitor well is completed. Initial sample results show only a 0.2 PPB C-8 level. Analysis of hydrology based on this result is still in progress.

F. Define total C-8 use and disposal mass balance at the using sites.

Responsibility - Washington Works - Mike McClusky, Roger Zipfel
Dordrecht Works - Charlie Muelder
Shimizu - Gary Herridge
Chambers Works - Al Morris
Major Customers - Bob Smith

Timing - 1Q93
Status - most sites have much of this data in hand. Need to complete balances.

G. Develop plan to reduce the landfilling of C-8 by 90%.

Responsibility - Roger Zipfel
Timing - 3Q/93
Status - Initial plans were to use depolymerization process to cosume waste polymer. Presently looking at cleaning up the site's waste polymer for sale into scrap markets. FEP effort is completed. Basic data for concept for coagulum recovery is nearly complete. Once concept is chosen additional basic data maybe needed for equipment sizing.

H. Develop plan to close the Letart Landfill

Responsibility - Walt Stewart,Roger Zipfel
Timing - 1993
Status - duPont will submit a closure permit application for the landfill in July, 1993. Closure plan will have the landfill operating through 1995. Plans for the handling of non-polymeric materials is in progress. Site will assist in developing a metals

reclamation effort.

I. Implement use of recovered C-8 in FEP plant

Responsibility - Roger Zipfel
Timing - 3Q93
Status - Current material is sent to 3M for purification prior to use in the fine powder/dispersion area. FEP maybe able to use current

quality material without 3M purification step.

J. Include C-8 abatement program in the site business plan.

Responsibility - Roger Zipfel
Timing - 12/92
Status - Current business plan will require modification.

to: CDCIL1::ISCDCVM2::RITCHERL

Once concept is chosen additional basic data maybe needed for equipment sizing.

H. Develop plan to close the Letart Landfill

Responsibility - Walt Stewart,Roger Zipfel
Timing - 1993
Status - duPont will submit a closure permit application for the

CC: POLYMERS OCCUPATIONAL HEALTH
COPY LIST

DU PONT POLYMERS
Achieving greatness through people

JUNE 11, 1991

TO: POLYMERS OCCUPATIONAL HEALTH SITE CONTACTS

FROM: AMY S. BERG
Amy S Berg

AELs - ACCEPTABLE EXPOSURE LIMITS

The following changes were made in the AEL list at the June meeting. Please replace the corresponding pages in your AEL list with the attached.

Ammonium Perfluoro-octanoate (C-8) (Polymers) [3825-26-1]	CEGw = 1 ug/L.
DPX-E9636 (Used in Titus\ Herbicide) (AG) [122931-48-0]	AEL = 5 mg/m3 (8- and 12-hour TWA), total dust.
Propylene Glycol Monomethyl Ether Acetate (IMG) [108-65-6]	AEL = 10 ppm (8- and 12-hour TWA).
RODA (Chemicals) [2479-46-1]	AEL = 0.5 mg/m3 (8- and 12-hour TWA).
Siduron (AG) [1982-49-6]	AEL = 10 mg/m3 (8- and 12-hour TWA), total dust).

EXHIBIT 1 D

Hydrazine (Fibers). An AEL of 0.05 ppm (8-hour TWA), skin was established in 1990. When hydrazine came up for finalization, it was decided to look at the data once more. After reviewing these data, it was decided to reduce the AEL to 0.01 ppm (8- and 12-hour TWA), skin. These data will be part of an updated hazard determination letter that will be released on June 7, 1991.

Dimethylacetamide [127-19-5]	AEL = 10 ppm (12-hour TWA), skin.
HCFC-123 [306-83-2]	EEL = 1000 ppm (2-60 minutes) with a 2500 ppm 1-minute ceiling concentration.

RA1000001

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Note that you were mailed a complete new list in May. Any pages from old revisions or lists (with dates in the lower left corner earlier than May 15, 1991) should be discarded.

DUPONT

EXHIBIT 46

April 23, 1991

FOR DU PONT USE ONLY

ACCEPTABLE EXPOSURE LIMITS (AEL) LIST - PREFACE

AELs

AELs are exposure limits for chemicals (or for levels of physical agents) set by the Du Pont AEL Committee. AELs specify Time-Weighted Average (TWA) airborne concentrations, doses or biological limits which should not be exceeded, and applicable time periods.

AELs may be set to prevent health effects from exposures for full workshifts (e.g., 8-hour or 12-hour TWA); or to prevent effects from shorter period exposures such as irritation, narcosis, odor or nuisance (e.g., 15-minute TWA). As a general guide, excursions to which short-period AELs apply should occur no more than four times per shift and a recovery period of approximately 30 minutes is required between excursions. In addition, the corresponding full shift (8-hour or 12-hour) AELs should not be exceeded.

AELs are set by the Du Pont AEL Committee, which includes experts in toxicology, industrial hygiene, occupational medicine, pathology, and epidemiology. AELs are based on the best available information from industrial experience, animal studies, and controlled human studies. They are guidelines based on informed judgment, and are not fine limits between safe and dangerous concentrations. They are not for use as relative toxicity indexes, limits for continuous uninterrupted exposure, or proof or disproof of health effects. They should be interpreted and applied by appropriately qualified personnel. Specific questions or consequences of occasional excursions above an AEL should be addressed to the Safety, Health and Environmental Affairs (SHEA) Manager for your business or staff function. Du Pont Engineering Standard S-12-T, "Strategy for Workspace Sampling for Exposures to Chemicals", provides guidelines for evaluation of air sampling data.

An AEL is established in three basic steps. The first step is a request for an AEL by a staff or business function. The second is review of the available toxicity and human health data followed either by a recommendation for a provisional AEL or a recommendation for additional information (i.e., additional testing, or more complete test data from another company). An AEL is in effect but provisional for six months; it is then reviewed to become a final AEL in light of workplace experience and any new data. This review, the third step, concludes the process. However, AELs are updated every five years, or sooner if warranted by new data, by a special subcommittee appointed by the AEL Committee. If this update indicates new data are available that might result in a change in the AEL, the chemical is referred back to the AEL Committee for review.

EID097178

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COMMUNITY EXPOSURE GUIDELINES (CEGs)

CEGs are exposure guidelines that are expected to be without any effect to members of the community during continuous 24-hour a day exposure to a chemical or physical agent. CEGs may be recommended for air or water or for both. As with AELs, CEGs are based on the best available information from industrial experience, animal toxicity studies, controlled human exposure studies, and epidemiological findings. However, because of the variability of sensitivities of members of the community (e.g., the infirm, the old, the young, pregnant females, etc.), versus the healthy worker involved with an AEL, a larger uncertainty factor needs to be used in extrapolating these data to a CEG.

EMERGENCY EXPOSURE LIMITS (EELs)

EELs are set for emergency situations, such as a spill or accidental release of a chemical. They specify brief durations and concentrations from which escape is feasible without any escape-impairing or irreversible effects on health. EELs are only applicable to emergency situations where occurrence is expected to be rare in the lifetime of an individual.

OTHER SOURCES OF EXPOSURE LIMITS

AELs supplement any mandatory regulatory limits developed by national or local governmental agencies. The more stringent limit, either that developed by Du Pont or by the regulatory agency, shall apply.

The American Conference of Governmental Industrial Hygienists (ACGIH) annually publishes a booklet containing Threshold Limit Values (TLVs) for many chemical substances and physical agents. Also, the American Industrial Hygiene Association (AIHA) publishes Workplace Environmental Exposure Limits (WEELs) for some chemicals not found in the TLV booklet. ACGIH TLVs and AIHA WEELs should be used as guidelines for workplace exposures if no other more appropriate limit exists. If a staff or business function has some concern about the validity of a TLV or WEEL, then the AEL Committee should be asked to establish an AEL.

Other compilations of limits (e.g., American Society of Testing and Materials (ASTM) and American National Standards Institute (ANSI) should be used after consultation with your Safety, Health and Environmental Affairs Manager and with Haskell Laboratory.

FOR DU PONT USE ONLY

HAZARD DETERMINATION GUIDELINES

In Du Pont, hazard determination is defined in a corporate policy (1) quoted below:

When toxicologic and/or epidemiologic data indicate that a chemical might present a carcinogenic, reproductive, developmental, or mutagenic hazard, any staff or business function which proposes to initiate the hazard determination procedure shall inform other interested staff and business functions before issuing a formal request for such determination. Following receipt of the request, the Director of Haskell Laboratory and the Corporate Medical Director shall evaluate the data, and after review by the Vice President of Safety, Health and Environmental Affairs, shall discuss their evaluation with the involved staff and/or business functions. This discussion should cover the extent of knowledge about the hazard associated with the chemical and should also give an indication about the potency of the chemical. The Director of Haskell Laboratory and the Corporate Medical Director will confirm the results of the discussion by letter to the appropriate SHEA manager(s) or their representative.

Carcinogens, developmental and reproductive toxins, and mutagens are defined as follows:

Carcinogen - A substance or agent with the potential to produce or incite cancer. Potency is determined by consideration of the following factors:

- Amount of chemical (dose) required to produce the effect
- Route of exposure
- Type of tumor(s), site, benign or malignant
- Number of animal species affected
- Tumor incidence
- Time to tumor formation
- Metabolism
- Genotoxic effects
- Other factors such as hormonal status, target organ for non-carcinogenic lesions, etc.

Substances or agents considered potent are identified on the AEL List by a capital letter C; less potent substances or agents are identified by a small letter c; substances or agents not considered to be carcinogens are identified by a C in parentheses, e.g., (C).

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(1) "Guidelines: Control of Carcinogenic, Reproductive, Developmental, and Mutagenic Risks Posed by Chemicals Made or Used within Du Pont". ELC Corporate Policy and Guidelines, IIC (February 1990).

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Developmental Toxin - An agent with the potential to interfere with the development of an individual while in utero or after birth.

Potency is determined by the Developmental Hazard Index (DHI) which is the ratio of the minimum dose toxic to the mother and the minimum dose toxic to the conceptus. Substances or agents with DHIs of greater than 5 are considered potent and are identified on the AEL List by a capital letter D; DHIs of 3 to 5 indicate a less potent substance or agent and are identified on the AEL List by a small letter d; substances or agents with a DHI of less than 3 are not considered developmental toxins and are identified on the AEL List by a D in parentheses, e.g., (D).

Reproductive Toxin - An agent with the potential to affect adversely the reproductive process of adult males and/or females.

Potency is determined as follows:

- Reproductive toxicity occurred at a dose level considerably below that resulting in other signs of toxicity. These substances or agents are considered potent and are indicated on the AEL List by a capital letter R. Male or female will also be indicated if reproductive toxicity occurred only in one sex.

- Reproductive toxicity occurred at a dose level at or just below that resulting in other signs of toxicity. These substances or agents are considered less potent and are identified on the AEL List by a small letter r. Male or female will also be indicated if reproductive toxicity occurred only in one sex.

- If reproductive toxicity occurred, but only at a dose level considerably greater than that resulting in other signs of toxicity, these substances or agents are not considered reproductive toxins and are identified on the AEL List by an R in parentheses, e.g., (R).

Mutagen - A mutagen is an agent with the potential to cause permanent heritable damage in germ (reproductive) cells of exposed individuals. A substance is identified as a mutagen if it is:

- A proven germ cell mutagen,
- Positive in a mammalian in vivo germ cell assay for gene mutations or chromosome aberrations, and/or
- Positive in a mammalian in vivo somatic (non-reproductive) cell assay for gene mutations or chromosome aberrations, and, in addition, the substance is either positive in a mammalian in vivo germ cell assay for DNA damage and repair, or is identified on the AEL List as a reproductive toxin.

Potency is determined by evaluating the following:

- The experimental design and route of administration.
- The dose required to produce genotoxicity.
- The magnitude of the genotoxic response and the presence of a dose-response relationship.
- The general concordance of positive findings among different germ cell genotoxicity assays (if known).
- The genetic endpoint assessed (gene mutations, chromosome aberrations, DNA repair).

Potent mutagens are identified on the AEL List by a capital letter M whereas less potent mutagens receive a small letter m. Agents not considered to be mutagens are identified by a capital letter M in parentheses, e.g., (M).

LIMITS FOR NON-FIBROUS AEROSOLS

The particle size distribution of inhaled material plays a major role in how much and where material is deposited within the respiratory tract. In general, particles having a mass median aerodynamic diameter greater than 30 micrometers are non-respirable. Respirable-size particles are typically defined as particles with a mass median aerodynamic diameter of less than or equal to 3 micrometers. Particles between 30 and 5 micrometers are deposited in the upper respiratory tract (nose) and do not pose a significant hazard to the airway and gas exchange region of the lung. Respirable particles which can deposit in the gas exchange region (< 1 micrometer) can interfere with oxygen transfer or pass directly into the blood. Some AELs for aerosols pertain only to the respirable fraction and these would be so designated on the AEL list. Compliance with respirable fraction AELs is determined from the fraction of aerosol passing a size selector. Thus, when sampling for particulate in air, the particle size (respirable fraction) must be established as follows:

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RESPIRABLE AEROSOL DEFINITION

Some AELs for aerosols pertain only to the respirable fraction, i.e., that portion of the aerosol which is small enough to reach the lower respiratory tract. Compliance with these AELs should be determined from the fraction of aerosol passing a size selector with the following characteristics (2).

Aerodynamic Diameter (microns)	Percent Passing Selector
$\leq$ 2.0	90
2.5	75
3.5	50
5.0	25
10.0	0

The AEL for particulates is generally expressed as milligrams per cubic meter (mg/m^3) total particulate. Respirable fractions are routinely assumed to be not more than 1/2 of the total particulate limit. Limits are established on a respirable fraction basis only when the particulate poses a significant hazard to the airway gas exchange region of the lung.

LIMITS FOR FIBERS

Fibrous dusts present a special hazard because the physical properties of dust (length versus width of the particle) impart special aerodynamic and, as a result, toxicologic characteristics.

A fiber is defined as a particle having an aspect ratio (length:width) greater than 3. In addition, the fiber must be of respirable size.

Until recently, a mass standard was used for quantification of fiber exposure. However, it has now been demonstrated that the utilization of gravimetric concentrations for comparing the relative toxicities of different fiber types is misleading. For this reason, fiber concentrations are usually reported as fibers/cc.

The AEL Committee has established an upper limit of 2 fibers/cc which incorporates advancing understanding of the biological consequences of deposition of respirable fibers.

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(2) AIHA Aerosol Technology Committee: Interim Guide for Respirable Mass Sampling, Am. Ind. Hyg. Assoc. J., 31(2):133 (1970).

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NUISANCE DUST LIMITS

Nuisance dusts are those that appear to have no biological effects at exposure levels that do not overload lung clearance mechanisms. Total particulate concentration for nuisance dusts should not exceed 10 mg/m^3. This limit is set to prevent reduced visibility, to prevent deposits in the eyes, ears and nasal passages, and to prevent injury to the skin or mucous membranes caused by chemical contact or by the mechanical process of cleansing. Respirable concentrations of nuisance dusts usually do not exceed 5 mg/m^3. This limit for nuisance respirable particulate should 1) protect the architecture of the air space, 2) prevent the formation of significant amounts of collagen (scar tissue), and 3) protect against the development of non-reversible particle-induced lung injury.

EXPLANATION OF AEL LIST

Chemical [CAS Registry Number]

The more common chemical name used within Du Pont and its Chemical Abstracts Service (CAS) Registry Number are given.

AEL

AELs for particulates are expressed as mg/m^3 and apply to actual site temperature and pressure conditions. Sampled air volumes should not be converted to 760 mm Hg and 25°C when calculating measured mg/m^3 concentrations for comparisons with AELs.

AELs for gases and vapors are expressed as parts per million (ppm by volume) at 760 mm Hg and 25°C. Measured ppm air concentrations should be compared with these limits under comparable temperature and pressure conditions.

Biological limits are the allowable concentration of a chemical or its metabolites found in a body specimen (e.g., blood or urine). The units may vary depending on the body specimen used (e.g., a blood limit would be expressed as ug of chemical per 100 g (dL) of blood).

REMARKS

This column contains additional information such as AEL averaging time (e.g., 8-hour TWA), regulatory classifications (e.g., OSHA Regulated), other appropriate limits (e.g., TLV or WEEL), particulate information (e.g., total dust), and any skin notation.

The skin notation indicates that the chemical may be absorbed through the skin or mucous membranes in toxicologically significant amounts. This notation implies that measures must be taken to minimize cutaneous contact. Corrosive chemicals are not identified by this notation.

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DATE/STATUS

Provides the year an AEL was initially finalized or most recently updated or indicates that an AEL is still provisional (P) and the year it was made provisional. AELs are updated every five years or sooner if warranted by new data. The Secretary of the AEL Committee maintains a file showing the history of the AELs; i.e., when the AEL was established, when updates occurred, etc.

ELC GUIDELINES

The symbols used in this column are defined below. If you have any question about the significance of any symbol, contact your Safety, Health and Environmental Affairs Manager.

The capital letters "C", "R", "D", and "M" identify chemicals that have undergone a hazard determination and a decision has been made that a special annual employee communication is REQUIRED and must be documented (S&OH Guideline 9.2) concerning the chemical's carcinogenic, reproductive, developmental, or mutagenic hazard. The Special Procedure dictated by ELC Policy IC applies. These chemicals are considered potent.

The small letters "c", "r", "d", and "m" identify chemicals that have undergone a hazard determination and a decision has been made that a special annual employee communication is NOT REQUIRED, provided that (1) the results of the hazard determination are included with the normal toxicity information available to employees about chemicals in their workplace, and (2) upon completion of the hazard determination, employees shall be notified of the results of that hazard determination. The Special Procedure dictated by ELC Policy IC applies. These chemicals are considered less potent.

Parentheses (C), (R), (D), and (M) identify chemicals that have undergone a hazard determination and a decision has been made that no hazard exists. The Special Procedure dictated by ELC Policy IC does not apply.

NEW ENTRIES OR CHANGES SINCE LAST ISSUE OF THE LIST

The "†" symbol in the far left column indicates a new entry on the list or a change has been made since its last issue.

Richard C. Graham
AEL8.10
April 22, 1991

EID097185

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ACCEPTABLE EXPOSURE LIMITS (AEL) LIST - PREFACE

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AELs may be set to prevent health effects from exposures for full workshifts (8- or 12-hour TWA); or to prevent effects from shorter period exposures such as irritation, narcosis, odor or nuisance (15-minute TWA). As a general guide, excursions to which short-period AELs apply should occur no more than four times per shift and a recovery period of approximately 60 minutes is required between excursions. In addition, the corresponding full shift (8- or 12-hour) AELs should not be exceeded.

AELs are set by the DuPont AEL Committee, which includes experts in toxicology, industrial hygiene, occupational medicine, pathology, and epidemiology. AELs are based on the best available information from industrial experience, animal studies, and controlled human studies. They are guidelines based on informed judgment, and are not fine limits between safe and dangerous concentrations. They are not for use as relative toxicity indexes, limits for continuous uninterrupted exposure, or proof or disproof of health effects. They should be interpreted and applied by appropriately qualified personnel. Specific questions or consequences of occasional excursions above an AEL should be addressed to the Safety, Health and Environmental Affairs (SHEA) Manager for your business or staff function. DuPont Engineering Standard S-12-T, provides guidelines for evaluation of air sampling data.

An AEL is established in three steps. The first step is a request for an AEL by a business or staff function. The second is a review of the available toxicity and human health data followed either by a recommendation for a provisional AEL or a recommendation for additional information (i.e., additional testing, or more complete test data from another company). An AEL is provisional, typically for six months. At the end of this provisional period, the AEL is reviewed again in light of any new data, before it is declared to be a final AEL. This review, the third step, concludes the process. An AEL goes into effect once it becomes final. AELs are updated every five years, or sooner if warranted by new data, by a special subcommittee appointed by the AEL Committee. If this update indicates new data are available that might result in a change in the AEL, the chemical is referred back to the AEL Committee for review.

Note: Material Safety Data Sheets for the chemical or mixtures containing the chemical must be revised within 90 days of the provisional AEL being established.

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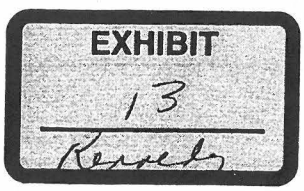

COMMUNITY EXPOSURE GUIDELINES (CEGs)

A Community Exposure Guideline (CEG) is an exposure guideline established by Haskell Laboratory. The CEG assumes a 24-hour lifetime exposure by all, including the most sensitive individuals, in an exposed community population. Exposure above the CEG will not necessarily result in any adverse effects. Where data indicates that the CEG may be approached or exceeded, Haskell, the appropriate business, and Legal will evaluate what action, if any, should be taken. It is the Company's intent to maintain exposure below the CEG.

CEGs may be recommended for air or water or for both. As with AELs, CEGs are based on the best available information from industrial experience, animal toxicity studies, controlled human exposure studies, and epidemiological findings. However, because of the variability of sensitivities of members of the community (e.g., the infirm, the old, the young, pregnant females, etc.), versus the healthy worker involved with an AEL, a larger uncertainty factor needs to be used in extrapolating these data to a CEG.

EMERGENCY EXPOSURE LIMITS (EELs)

EELs are set for emergency situations, such as a pill or accidental release of a chemical. They specify brief durations and concentrations from which escape is feasible without any escape-impairing or irreversible effects on health. EELs are only applicable to emergency situations where occurrence is expected to be rare in the lifetime of an individual.

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Other compilations of limits (e.g., American Society of Testing and Materials (ASTM) and American National Standards Institute (ANSI) should be used after consultation with your SHEA Manager and with Haskell Laboratory.

FOR DUPONT USE ONLY

HAZARD DETERMINATION GUIDELINES

In DuPont, hazard determination is defined in a corporate policy (1) quoted below:

When toxicologic and/or epidemiologic data indicate that a chemical might present a carcinogenic, reproductive, developmental, or germ-cell mutagenic hazard, any business or staff function which proposes to initiate the hazard determination procedure shall inform other interested businesses and staff functions before issuing a formal request for such determination. Following receipt of the request, the Director of Haskell Laboratory and Corporate Medical shall evaluate the data, and after review by the Vice President of Safety, Health and Environment shall discuss their evaluation with the involved businesses and/or staff functions. This discussion should cover the extent of knowledge about the hazard associated with the chemical and should also give an indication about the potency of the chemical. The Director of Haskell Laboratory and Corporate Medical will confirm the results of the discussion by letter to the appropriate SHEA manager(s) or their representative.

Carcinogens, developmental and reproductive toxins, and germ-cell mutagens are defined as follows:

Carcinogen - A substance or agent with the potential to produce or incite cancer. For The Carcinogen Classification System, a weight of evidence analysis is used with all the following factors considered in the evaluation (NOTE: These factors are not listed with any rank or priority). The categories included in the Carcinogen Classification System are found on the next page.

- Amount of chemical (dose) required to produce the effect
- Route of exposure relative to potential human experience
- Type of tumor(s), site of tumors, and whether the tumors are benign or malignant
- Number of animal species affected
- Tumor incidence
- Time to tumor formation
- Genotoxicity data
- Mechanistic data
- Pharacokinetics and metabolism
- Structure Activity Relationship
- Epidemiologic studies

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(1) "Guidelines: Control of Carcinogenic, Reproductive, Developmental, and Germ-Cell Mutagenic Risks Posed by Chemicals Made or Used within DuPont". ELC Corporate Policy and Guidelines, IIC (September 1991).

CARCINOGEN CLASSIFICATION SYSTEM

C-H KNOWN HUMAN CARCINOGEN

Substances which are known to be carcinogenic in humans. There is sufficient evidence, based on epidemiology data, to establish a causal association between exposure to the substance and the development of cancer.

C-A PROBABLE HUMAN CARCINOGEN (POTENT ANIMAL CARCINOGEN)

There is sufficient evidence in one or more adequately conducted studies that the substance is clearly carcinogenic in experimental animals.

There are no epidemiology data available or the existing epidemiology data are conflicting or limited/insufficient to establish a causal association between human exposure and the development of cancer.

c POSSIBLE HUMAN CARCINOGEN (WEAK ANIMAL CARCINOGEN OR LIMITED EVIDENCE IN ANIMALS)

There is some or limited evidence that the substance is carcinogenic in experimental animals.

There are no epidemiology data available or the existing epidemiology data are conflicting or limited/insufficient to establish a causal association between human exposure and the development of cancer.

(c) NOT LIKELY TO BE A HUMAN CARCINOGEN (ANIMAL CARCINOGEN UNLIKELY TO HAVE HUMAN RELEVANCE)

There is sufficient or limited evidence in experimental animal studies that the substance is carcinogenic at high dose levels (may have exceeded the MTD), by routes of administration, in tissues, or by mechanisms that are not considered relevant to potential human exposure.

(NC) NOT CONSIDERED TO BE A CARCINOGENIC HAZARD TO HUMANS (LACK OF EVIDENCE OF CARCINOGENICITY)

There is evidence from an adequately conducted experimental animal study showing a lack of carcinogenicity.

If any epidemiology evidence exists, it supports the conclusion that there is no known association between exposure and and increase in cancer risk to humans.

Developmental Toxin - An agent with the potential to interfere with the development of an individual while in utero or after birth.

Potency is determined by the Developmental Hazard Index (DHI) which is the ratio of the minimum dose toxic to the mother and the minimum dose toxic to the conceptus. Substances or agents with DHIs of greater than 5 are considered potent and are identified on the AEL List by a capital letter D; DHIs of 3 to 5 indicate a less potent substance or agent and are identified on the AEL List by a small letter d; substances or agents with a DHI of less than 3 are not considered developmental toxins and are identified on the AEL List by a D in parentheses, e.g., (D).

Reproductive Toxin - An agent with the potential to affect adversely the reproductive process of adult males and/or females.

Potency is determined as follows:

- Reproductive toxicity occurred at a dose level considerably below that resulting in other signs of toxicity. These substances or agents are considered potent and are indicated on the AEL List by a capital letter R. Male or femal- will also be indicated if reproductive toxicity occurred only in one sex.

- Reproductive toxicity occurred at a dose level at or just below that resulting in other signs of toxicity. These substances or agents are considered less potent and are identified on the AEL List by a small letter r. Male or female will also be indicated if reproductive toxicity occurred only in one sex.

- If reproductive toxicity occurred, but only at a dose level considerably greater than that resulting in other signs of toxicity, these substances or agents are not considered reproductive toxins and are identified on the AEL List by an R in parentheses, e.g., (R).

Germ-Cell Mutagen - A genotoxic agent with the potential to cause permanent heritable damage in germ (reproductive) cells of exposed individuals. A substance is identified as a mutagen if it is:

- A proven human germ-cell mutagen,
- Positive in a mammalian in vivo germ-cell assay for gene mutations or chromosome aberrations, or
- Positive in a mammalian in vivo somatic (non-reproductive) cell assay for gene mutations or chromosome aberrations, and, in addition, the substance is either positive in a mammalian in vivo germ-cell assay for DNA damage and repair, or is identified on the AEL List as a reproductive toxin.

In evaluating experimental studies in mammals, the following factors are considered:

- The experimental design and route of administration.
- The dose required to produce genotoxicity.
- The magnitude of the genotoxic response and the presence of a dose-response relationship.
- The general concordance of positive findings among different germ-cell genotoxicity assays (if known).
- The genetic endpoint assessed (gene mutations, chromosome aberrations, DNA repair).

Potent mutagens are identified on the AEL List by a capital letter M whereas less potent mutagens receive a small letter m. Agents not considered to be mutagens are identified by a capital letter M in parentheses, e.g., (M).

Potent germ-cell mutagen categorization M is primarily applied to:

- Proven human germ-cell mutagens, or
- Experimental mammalian germ-cell mutagens with a strong evidence of causing genotoxic damage in humans.

In general, validated germ-cell mutagens in experimental mammals receive a small letter m.

Although genotoxic agents also affect somatic (non-reproductive) cells, the guidelines described here address only genetic damage to germ cells. Genotoxic effects on somatic cells are usually addressed in carcinogen hazard determinations (see page 3).

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LIMITS FOR NON-FIBROUS AEROSOLS

The particle size distribution of inhaled material plays a major role in how much and where material is deposited within the respiratory tract. In general, particles having a mass median aerodynamic diameter greater than 30 micrometers are non-respirable. Respirable-size particles are typically defined as particles with a mass median aerodynamic diameter of less than or equal to 3 micrometers. Particles between 30 and 5 micrometers are deposited in the upper respiratory tract (nose) and do not pose a significant hazard to the airway and gas exchange region of the lung. Respirable particles which can deposit in the gas exchange region ($\leq$ 1 micrometer) can interfere with oxygen transfer or pass directly into the blood. Some AELs for aerosols pertain only to the respirable fraction and these would be so designated on the AEL list. Compliance with respirable fraction AELs is determined from the fraction of aerosol passing a size selector. Thus, when sampling for particulate in air, the particle size (respirable fraction) must be established as follows:

RESPIRABLE AEROSOL DEFINITION

Some AELs for aerosols pertain only to the respirable fraction, i.e., that portion of the aerosol which is small enough to reach the lower respiratory tract. Compliance with these AELs should be determined from the fraction of aerosol passing a size selector with the following characteristics (2).

Aerodynamic Diameter (microns)	Percent Passing Selector
$\leq$ 2.0	90
2.5	75
3.5	50
5.0	25
10.0	0

The AEL for particulates is generally expressed as milligrams per cubic meter (mg/m^3) total particulate. Respirable fractions are routinely assumed to be not more than 1/2 of the total particulate limit. Limits are established on a respirable fraction basis only when the particulate poses a significant hazard to the airway gas exchange region of the lung.

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(2) AIHA Aerosol Technology Committee: Interim Guide for Respirable Mass Sampling, Am. Ind. Hyg. Assoc. J., 31(2):133 (1970).

LIMITS FOR FIBERS

Fibrous dusts present a special hazard because the physical properties of dust (length versus width of the particle) impart special aerodynamic and, as a result, toxicologic characteristics.

A fiber is defined as a particle having an aspect ratio (length:width) greater than 3. In addition, the fiber must be of respirable size. Respirable fibers are defined within DuPont as being less than 3 microns in diameter, greater than 5 microns in length, and having an aspect ratio of greater than 3:1.

Until recently, a mass standard was used for quantification of fiber exposure. However, it has now been demonstrated that the utilization of gravimetric concentrations for comparing the relative toxicities of different fiber types is misleading. For this reason, fiber concentrations are usually reported as fibers/cc.

The AEL Committee has established an upper limit of 2 fibers/cc which incorporates advancing understanding of the biological consequences of deposition of respirable fibers.

NUISANCE DUST LIMITS

Nuisance dusts are those that appear to have no biological effects at exposure levels that do not overload lung clearance mechanisms. Total particulate concentration for nuisance dusts should not exceed 10 mg/m³. This limit is set to prevent reduced visibility, to prevent deposits in the eyes, ears and nasal passages, and to prevent injury to the skin or mucous membranes caused by chemical contact or by the mechanical process of cleansing. Respirable concentrations of nuisance dusts usually do not exceed 5 mg/m³. This limit for nuisance respirable particulate should 1) protect the architecture of the air space, 2) prevent the formation of significant amounts of collagen (scar tissue), and 3) protect against the development of non-reversible particle-induced lung injury.

EXPLANATION OF AEL LIST

Chemical [CAS Registry Number]

The more common chemical name used within DuPont and its Chemical Abstracts Service (CAS) Registry Number are given.

AEL

AELs for particulates are expressed as mg/m^3 and apply to actual site temperature and pressure conditions. Sampled air volumes should not be converted to 760 mm Hg and 25°C when calculating measured mg/m^3 concentrations for comparisons with AELs.

AELs for gases and vapors are expressed as parts per million (ppm by volume) at 760 mm Hg and 25°C. Measured ppm air concentrations should be compared with these limits under comparable temperature and pressure conditions.

Biological limits are the allowable concentration of a chemical or its metabolites found in a body specimen (e.g., blood or urine). The units may vary depending on the body specimen used (e.g., a blood limit would be expressed as ug of chemical per 100 g (dL) of blood).

REMARKS

This column contains additional information such as AEL averaging time (e.g., 8-hour TWA), regulatory classifications (e.g., OSHA Regulated), other appropriate limits (e.g., TLV or WEEL), particulate information (e.g., total dust), and any skin notation.

The skin notation indicates that the chemical may be absorbed through the skin or mucous membranes in toxicologically significant amounts. This notation implies that measures must be taken to minimize cutaneous contact. Corrosive chemicals are not identified by this notation.

DATE/STATUS

Provides the year an AEL was initially finalized or most recently updated or indicates that an AEL is still provisional (P) and the year it was made provisional. AELs are updated every five years or sooner if warranted by new data. The Secretary of the AEL Committee maintains a file showing the history of the AELs; i.e., when the AEL was established, when updates occurred, etc.

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EXPLANATION OF AEL LIST (CONT'D)

ELC GUIDELINES

The symbols used in this column are defined below. If you have any question about the significance of any symbol, contact your SHEA Manager.

The capital letters "C-H", "C-A", "R", "D", and "M" identify chemicals that have undergone a hazard determination and a decision has been made that a special annual employee communication is REQUIRED and must be documented (S&OH Guideline 9.2) concerning the chemical's carcinogenic, reproductive, developmental, or germ-cell mutagenic hazard. The Special Procedure outlined in ELC Policy IC applies. These chemicals are considered potent.

The small letters "c", "r", "d", and "m" identify chemicals that have undergone a hazard determination and a decision has been made that a special annual employee communication is NOT REQUIRED, provided that (1) the results of the hazard determination are included with the normal toxicity information available to employees about chemicals in their workplace, and (2) upon completion of the hazard determination, employees shall be notified of the results of that hazard determination. The Special Procedure outlined in ELC Policy IC applies. These chemicals are considered less potent.

Parentheses (NC), (c), (R), (D), and (M) identify chemicals that have undergone a hazard determination and a decision has been made that no hazard exists. The Special Procedure outlined in ELC Policy IC does not apply.

NEW ENTRIES OR CHANGES SINCE LAST ISSUE OF THE LIST

The "†" symbol in the far left column indicates a new entry on the list or a change has been made since its last issue.

Richard C. Graham
June 17, 1994
AEL30.6

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Acetaldehyde [75-07-0]	25 ppm	8-hour TWA	1992	c 1986
Acetaminophen [103-90-2]	5 mg/m³	8-hour TWA, total dust	1990	
†Acetic Acid [64-19-7]	10 ppm	8- and 12-hour TWA	1992	
Acetone Cyanohydrin [75-86-5]	10 ppm	15-minute TWA, skin	1990	
Acravax® C - [110-30-5]	10 mg/m³	8-hour TWA	1990	
Acrolein [107-02-8]	0.1 ppm	8-hour TWA	1990	
†Acrylic Acid [79-10-7]	2 ppm	8- and 12-hour TWA	1993	(NC 1993)* (R 1993)* (D 1993)* (M 1993)*
†Acrylonitrile [107-13-1]	0.5 ppm 2 ppm	8- and 12-hour TWA, skin 15-minute TWA, skin OSHA Regulated Carcinogen, PEL - 2 ppm (8-hour TWA); 10 ppm (15-minute TWA), skin	1987	C-A 1986 (D 1986)* (R 1986)*
Adipic Acid [124-04-9]	10 mg/m³	8-hour TWA	1990	
Adiponitrile [111-69-3]	2 ppm	8-hour TWA	1990	
Alathon® (polyethylene) [9002-88-4]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1988	

* Substances or agents revieved according to ELC Guidelines and not considered to be a carcinogenic, developmental, reproductive, or germ-cell mutagenic hazard are indicated by the appropriate letter within parentheses, e.g., (NC), (c), (D), (R), or (M).

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Alkanol® XC Surfactant [68442-09-1]	0.05 mg/m³	8- and 12-hour TWA	1994	
Ally® Weed Killer		See Metsulfuron Methyl		
Aluminum Isopropoxide [555-31-7]	5 mg/m³	8-hour TWA	1990	
Aluminum Stearate [637-12-7]	10 mg/m³	8-hour TWA	1991	
p-Aminoazobenzene [60-09-3]	1 mg/m³	8- and 12-hour TWA	1990	c 1983
2-Aminodiphenyl [90-41-5]	5 mg/m³	8- and 12-hour TWA, skin	1990	c 1983
2-Aminodiphenyl Hydrochloride [2185-92-4]	5 mg/m³	8- and 12-hour TWA, skin	1990	c 1983
4-Aminodiphenyl [92-67-1]	----	OSHA Regulated Carcinogen - See AEL Documentation for control strategy	1990	
†3-Amino-1,2,4-triazole (Amitrole) [61-82-5]	0.2 mg/m³	8-hour TWA	1990	c 1984 (D 1984)* (R 1984)*
Ammonia [7664-41-7]	25 ppm	8- and 12-hour TWA	1987	
Ammonium Bisulfate [7803-63-6]	1 mg/m³	8- and 12-hour TWA	1989	
†Ammonium Dichromate [7789-09-5]	0.01 mg/m³	8- and 12-hour TWA, as chromium ELC Reclassification Pending	P1994	c 1986

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Ammonium Nitrate [6484-52-2]	10 mg/m³	8-hour TWA	1990	
Ammonium Perfluorononanoate [4149-60-4]	0.01 mg/m³	8-hour TWA	1991	
†Ammonium Perfluorooctanoate [3825-26-1]	0.01 mg/m³	8-hour TWA, skin	1993	c 1988 (D 1988)*
†Ammonium Persulfate [7727-54-0]	5 mg/m³	8-hour TWA	1992	
Amorphous Silica		See Silica, Amorphous		
Aniline [62-53-3]	2 ppm	8- and 12-hour TWA, skin	1988	c 1990 (D 1990)*
Aniline Hydrochloride [142-04-1]	2 ppm	8- and 12-hour TWA, skin	1990	c 1990 (D 1990)*
†o-Anisidine [90-04-0]	0.5 mg/m³	8-hour TWA, skin	1990	C-A 1979
†o-Anisidine Hydrochloride [134-29-2]	0.5 mg/m³	8-hour TWA, skin	1990	C-A 1979
Anisole [100-66-3]	5 ppm	8-hour TWA	1990	
†Antimony Trioxide [1309-64-4]	0.2 mg/m³	8-hour TWA, as antimony	1990	C-A 1986
Antioxidant CAO-5 [119-47-1]	5 mg/m³	8-hour TWA	1990	
†Armostat 310 [61791-44-4]	5 mg/m³	8-hour TWA	1992	

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Aromatic 100 [64742-95-6]	50 ppm	8-hour TWA	1991	
†Aromatic 150 [64742-94-5]	100 ppm	8-hour TWA	1991	
Asana® Insecticide		See Esfenvalerate		
†Asbestos (Amosite, chrysotile, tremolite) [1332-21-4]	0.2 fibers /cc	8- and 12-hour TWA; fibers > 5 u long; Engineering Standard S4T applies; OSHA Regulated, PEL = 0.2 fibers/cc	1992	C-H 1986
Assure® Herbicide		See Quizalofop Ethyl		
Atrazine [1912-24-9]	0.5 mg/m³	8- and 12-hour TWA	1987	c 1987 (D 1987)* (R 1987)*
rium Chloride [10361-37-2]	0.8 mg/m³	8-hour TWA	1990	
Barium Sulfate [7727-43-7]	10 mg/m³	8-hour TWA	1990	
Barium Telomer B Sulfonic Acid	1 mg/m³	8- and 12-hour TWA, skin	1990	
Benomyl [17804-35-2]	5 mg/m³	8- and 12-hour TWA, total dust	1991	c 1991 r 1991(males) (D 1991)* (M 1991)*
Bensulfuron Methyl (Used in Londax® Herbicide) (INF-5384) [83055-99-6]	10 mg/m³	8- and 12-hour TWA	1988	
Benzaldehyde [100-52-7]	2 ppm	8-hour TWA	1989	

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WJB000015

June 17, 1994

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Benzene [71-43-2]	1 ppm 5 ppm	8- and 12-hour TWA 15-minute TWA OSHA PEL = 1 ppm	1989	C-H 1988 (D 1988)*
Benzoflex® S-312 [4196-89-8]	10 mg/m³	8-hour TWA	1990	
†Benzoic Acid [65-85-0]	10 mg/m³	8-hour TWA, particulate	1992	
Benzyl Alcohol [100-51-6]	10 ppm	8-hour TWA	1990	
†Benzyl Chloride [100-44-7]	1 ppm	8-hour TWA	1992	c 1986 (D 1986)*
†Benzyl Chloride Residue (BCR)	1 ppm	8-hour TWA See documentation for composition	1992	c 1986 (D 1986)*
Benzyltriphenyl-phosphonium Chloride [1100-88-5]	0.1 mg/m³	8- and 12-hour TWA	1990	
†Biphenyl [92-52-4]	0.2 ppm	8- and 12-hour TWA	1992	
Biphenyl Ether [101-84-8]	1 ppm	8- and 12-hour TWA	1988	
†Bisphenol A [80-05-7]	5 mg/m³	8-hour TWA	1992	
Bladex® Herbicide		See Cyanazine		
†Boric Acid [10043-35-3]	5 mg/m³	8- and 12-hour TWA, total dust	1994	
†Bromacil [314-40-9]	10 mg/m³	8- and 12-hour TWA	1988	(c 1987)* (D 1987)* (R 1987)*

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* Substances or agents reviewed according to ELC Guidelines and not considered to be a carcinogenic, developmental, reproductive, or germ-cell mutagenic hazard are indicated by the appropriate letter within parentheses, e.g., (NC), (c), (D), (R), or (M).

WJB000016

June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
beta-Bromoethyl-benzene [103-63-9]	1 ppm	8-hour TWA	1990	
p-Bromofluoro-benzene [460-00-4]	10 ppm	8- and 12-hour TWA	1989	
Bromotrifluoro-ethylene [598-73-2]	0.5 ppm	8-hour TWA	1990	
†1,3-Butadiene [106-99-0]	2 ppm	8- and 12-hour TWA	1991	C-A 1990 (D 1990)*
1,4-Butanediol [110-63-4]	30 ppm	8- and 12-hour TWA	1990	
n-Butanol [71-36-3]	25 ppm 50 ppm	8-hour TWA 15-minute TWA	1990	
2-Butanone		See Methyl Ethyl Ketone		
2-Butoxyethanol (Butyl Cellosolve®, Dowanol® EB) [111-76-2]	10 ppm	8-hour TWA, skin	1990	(D 1985)* (R 1985)*
2-Butoxyethyl Acetate [112-07-2]	20 ppm	8-hour TWA, skin	1990	
†n-Butyl Acrylate [141-32-2]	5 ppm	8- and 12-hour TWA, skin	1992	
p-tert-Butyl-benzoic Acid [98-73-7]	0.2 mg/m³ 0.6 mg/m³	8-hour TWA, skin 15-minute TWA, skin	1988	R 1983

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Butyl Benzyl Phthalate [85-68-7]	5 mg/m³	8-hour TWA	1988	
†Butyl Carbitol® (2-(2-Butoxyethoxy) ethanol) [112-34-5]	5 ppm	8-hour TWA	1992	
4-tert-Butylcatechol [98-29-3]	20 mg/m³ 10 mg/m³	8-hour TWA 12-hour TWA	1990	
Butyl Cellosolve®		See 2-Butoxyethanol		
Butyl Cellosolve® Acetate		See 2-Butoxyethyl Acetate		
†n-Butyl Chloride [109-69-3]	10 ppm	8-hour TWA	1992	
n-Butyl Isocyanate [111-36-4]	0.01 ppm 0.02 ppm	8- and 12-hour TWA 20-minute TWA	1992	
t-Butyl Isocyanide [7188-38-7]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
iso-Butyl Methacrylate [97-86-9]	100 ppm	8-hour TWA	1990	
n-Butyl Methacrylate [97-88-1]	100 ppm	8-hour TWA	1990	
tert-Butylurea [1118-12-3]	10 mg/m³	8-hour TWA	1990	
Butyraldehyde [123-72-8]	20 ppm	8- and 12-hour TWA	1990	
Cab-O-Sil® Amorphous Silica		See Silica, Amorphous		

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Cadmium and Cadmium Compounds which include:	0.01 mg/m³	8-hour TWA, as cadmium	1990	C-H 1990 (D 1990)* (R 1990)*
Cadmium metal (fume or dust) [7440-43-9] Cadmium borate [51222-60-7] Cadmium bromide [7789-42-6] Cadmium chloride [10108-64-2] Cadmium oxide [1306-19-0] Cadmium stannate [No CAS Number], and				
†Cadmium Pigments which include:			1987	C-H 1987
Cadmium Selenide [1306-24-7] Cadmium Sulfide [1306-23-6] Cadmium Sulfoselenide [12626-36-7]				
†Calcium Chloride [10043-52-4]	7 mg/m³	8- and 12-hour TWA	1992	
Calcium Fluoride [7789-75-5]	5 mg/m³	8-hour TWA	1990	
†Calcium Nitrate Tetrahydrate [13477-34-4]	5 mg/m³	8-hour TWA, total dust	1992	
Carbitol® Acetate [112-15-2]	10 ppm	8- and 12-hour TWA	1988	
2-Carbomethoxy-benzenesulfonamide		See IND-5803		
Carbon Black [1333-86-4]	3.5 mg/m³	8-hour TWA, Polynuclear aromatic hydrocarbon (PAHs) content <0.1%	1990	
Carbon Disulfide [75-15-0]	10 ppm	8- and 12-hour TWA, skin	1989	

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Carbon Fibers [Polyacrylo-nitrile (PAN) based or Du Pont pitch-based carbon fibers containing no PAHs	1 fiber/cc	8-hour TWA, Respirable fibers, fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio > 3:1	P1989	See 11-20-89 Hazard Det. Letter - Data not sufficent for classification
	3.5 mg/m³	Non-fibrous particulate (under review)		
Carbon Monoxide [630-08-0]	50 ppm 25 ppm	8-hour TWA 12-hour TWA	1986	
Carbon Tetrachloride [56-23-5]	5 ppm	8- and 12-hour TWA, skin	1990	c 1984 (D 1984)*
Carbonyl Sulfide [463-58-1]	2 ppm	8-hour TWA	P1990	
Cellosolve®		See 2-Ethoxyethanol		
Cellosolve® Acetate		See 2-Ethoxyethyl Acetate		
Ceramic Fibers		See Refractory Aluminum Silicate Ceramic Fibers		
CFC-113a (1,1,1-Trichloro-2,2,2-trifluoroethane) [354-58-5]	1000 ppm	8- and 12-hour TWA	1991	
CFC-114a (1,1-Dichloro-1,2,2,2-tetrafluoroethane) [374-07-2]	1000 ppm	8- and 12-hour TWA	1991	
Chloral [75-87-6]	10 ppb	8-hour TWA	1991	
†Chlorethoxyfos (Used in Fortress® Insecticide) [54593-83-8]	0.05 mg/m³ (4 ppb)	8-hour TWA, skin	1990	

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Chlorimuron Ethyl (Used in Classic® Herbicide) (DPX-P6025) [90982-32-4]	10 mg/m³ 5 mg/m³	8- and 12-hour TWA, total dust 8- and 12-hour TWA, respirable dust	1992	
†Chlorine [7782-50-5]	0.5 ppm 1 ppm	8- and 12-hour TWA 15-minute TWA	P1994	
m-Chloroaniline [108-42-9]	0.5 ppm	8- and 12-hour TWA, skin	1988	
o-Chloroaniline [95-51-2]	2 ppm	8- and 12-hour TWA, skin	1988	
p-Chloroaniline [106-47-8]	0.5 mg/m³ 0.3 mg/m³	8-hour TWA, skin 12-hour TWA, skin	1988	c 1988 (R 1988)*
†Chlorobenzene [108-90-7]	25 ppm	8- and 12-hour TWA	1986	(NC 1986)* (D 1986)*
1-Chloro-1,1-difluoroethane		See HCFC-142b		
Chlorodifluoromethane		See HCFC-22		
Chlorofluoromethane		See HCFC-31		
†Chloroform [67-66-3]	2 ppm	8- and 12-hour TWA	1993	c 1993 (D 1993)* (R 1993)* (M 1993)*
5-Chloro-2-methyl-3(2H)-isothiazolone [2682-20-4] mixture with 2-methyl-3(2H)-isothiazolone [26172-55-4] (Kathon® CG/ICP)	0.1 mg/m³	8- and 12-hour TWA	1991	
Chloroprene [126-99-8]	10 ppm	8- and 12-hour TWA	1988	
2-Chloro-1,1,1,2-tetrafluoroethane		See HCFC-124		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
2-Chloro-1,1,I-trifluoroethane		See HCFC-133a		
†Chlorothalonil (2,4,5,6-Tetra-chloro-1,3-benzenedicarbonitrile) [1897-45-6]	0.1 mg/m³	8-hour TWA	1992	
Chlorsulfuron (Used in Glean® Herbicide) [64902-72-3]	10 mg/m³	8- and 12-hour TWA	1990	
Chromic Acid		See Chromium Oxide (CrO_3)		
†Chromium Dioxide [12018-01-8]	0.1 mg/m³	8-hour TWA, as chromium	1993	(NC 1993)*
†Chromium Oxide (CrO_3) [1333-82-0]	0.05 mg/m³	8-hour TWA, as chromium ELC Reclassification Pending	1992	c 1986 (D 1986)*
Cinch® Herbicide		See Cinmethylin		
†Cinmethylin		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Classic® Herbicide		See Chlorimuron Ethyl		
†Coal Dust (<5% quartz)	2 mg/m³	8- and 12-hour TWA	1992	
†Cobalt Blue [1345-16-0 and 12572-27-4]	----	ELC Reclassification Pending	----	(C 1981)*
†m-Cresol [108-39-4]	5 ppm	8- and 12-hour TWA, skin	1992	
†o-Cresol [95-48-7]	5 ppm	8- and 12-hour TWA, skin	1992	
†p-Cresol [106-44-5]	5 ppm	8- and 12-hour TWA, skin	1992	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Cupric Chloride [1344-67-8]	2 mg/m³	8-hour TWA	1990	
Cupric Hydroxide [1344-69-0]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Cuprous Chloride [7758-89-6]	1.5 mg/m³	8-hour TWA	1990	
Cuprous Cyanide [544-92-3]	1.5 mg/m³	8-hour TWA	1990	
Curzate® Fungicide [57966-95-7]	10 mg/m³	8-hour TWA	1990	
Cyanazine (Used in Bladex® Herbicide) [21725-46-2]	0.5 mg/m³	8- and 12-hour TWA, skin, total dust	1991	c 1990 (D 1990)* (R 1990)* (M 1990)*
1,5,9-Cyclodo-decatriene [4904-61-4]	20 ppm	8- and 12-hour TWA	1990	
Cyclohexane [110-82-7]	150 ppm	12-hour TWA	1990	
Cyclohexyl Isocyanate [3173-53-3]	0.02 ppm	20-minute TWA	1990	
†1,5-Cyclooctadiene [111-78-4]	5 ppm	8- and 12-hour TWA	1992	
Cyclopentanone [120-92-3]	50 ppm	8-hour TWA	1990	
p-Cymene [99-87-6]	10 ppm	8-hour TWA	1990	
†1,1,1,2,2,3,4,5,5,5-Decafluoropentane		See HFC-43-10		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Delrin® (polyoxy-methylene) [25231-38-3]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1988	
†Dialifos [10311-84-9]	0.1 mg/m³	8-hour TWA, skin	1992	
1,2-Diamino-cyclohexane [694-83-7]	5 mg/m³	8- and 12-hour TWA	1991	
1,3-Diamino-cyclohexane [3385-21-5]	1 ppm	8-hour TWA	1990	
1,4-Diamino-cyclohexane [3114-70-3]	5 mg/m³	8- and 12-hour TWA	1991	
†o-Dianisidine [119-90-4]	0.01 mg/m³	8-hour TWA, skin	1990	C-A 1989
Dibasic Esters (DBE)	10 mg/m³ (1.5 ppm)	8-hour TWA See Documentation for composition	1990	
Dibromomethane [74-95-3]	10 ppm	8- and 12-hour TWA	1991	
N,N'-Dibutylhexa-methylenediamine [4835-11-4]	0.1 ppm	8-hour TWA	1990	
†Di(n-butyl) phthalate [84-74-2]	5 mg/m³	8-hour TWA	1992	d 1982 r 1982
†3,4-Dichloroaniline [95-76-1]	2 mg/m³	8- and 12-hour TWA, skin	1992	
o-Dichloro-benzene [95-50-1]	50 ppm	15-minute TWA	1988	
†2,3-Dichloro-1,3-butadiene [1653-19-6]	5 ppm	8- and 12-hour TWA	1992	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
3,4-Dichloro-butene-1 [760-23-6]	2 ppm	8-hour TWA	1985	
†1,3-Dichloro-butene-2 [926-57-8]	0.3 ppm	8- and 12-hour TWA	1993	
†1,4-Dichloro-butene-2 [764-41-0]	0.005 ppm	8- and 12-hour TWA, skin	P1994	C-A 1994 (D 1994)*
Dichloro(chloro-methyl)silane [1558-33-4]	5 ppm	15-minute TWA, as HCl	1990	
1,2-Dichloro-1,1-di-fluoroethane		See HCFC-132b		
Dichlorodimethyl-silane [75-78-5]	5 ppm	15-minute TWA, as HCl	1990	
1,1-Dichloro-1-fluoro ethane		See HCFC-141b		
Dichlorofluoro-methane		See HCFC-21		
1,1-Dichloro-1,2,2,2-tetra-fluoroethane		See CFC-114a		
2,2-Dichloro-1,1,1-tri-fluoroethane		See HCFC-123		
N,N-Diethylaniline [91-66-7]	5 ppm	8-hour TWA, skin	1986	
N,N-Diethyl-cyclohexylamine [91-65-6]	0.5 ppm	8-hour TWA	1990	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Diethylene Glycol [111-46-6]	100 ppm 10 mg/m³	8-hour TWA, vapor 8-hour TWA, aerosol	1990	
Di(2-ethylhexyl) phthalate [117-81-7]	5 mg/m³	8-hour TWA	1987	c 1982 d 1982 r 1982
1,1-Difluoroethane		See HFC-152a		
Difluoro(fluoro-sulfonyl)acetyl fluoride [677-67-8]	0.1 ppm	8- and 12-hour TWA	1991	
Difluoromethane		See HFC-32		
Diglyme [111-96-6]	1 ppm	8-hour TWA, skin	1988	D 1987 R 1987
6,7-Dihydro-2-methyl-5H-cyclopenta(d)pyrimidine ("Popcorn")[36274-29-0]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†Diisobutylcarbinol [108-82-7]	5 ppm	8-hour TWA	1992	
Diisobutylene Nitrosate [65152-04-7]	1 ppm	8-hour TWA	1989	
Dimethoxane [828-00-2]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†N,N-Dimethyl-acetamide [127-19-5]	10 ppm 10 ppm	8-hour TWA, skin 12-hour TWA, skin	1984 1991	(NC 1986)* (D 1986)* (R 1986)*
†N,N-Dimethyl-aniline [121-69-7]	2 ppm	8- and 12-hour TWA, skin	P1994	c 1994
†Dimethylcarbamoyl Chloride [79-44-7]	0.5 ppb	8-hour TWA	1990	C-A 1976

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
N,N-Dimethyl-ethanolamine [108-01-0]	2 ppm	8-hour TWA	1990	
†Dimethyl Ether [115-10-6]	1000 ppm	8- and 12-hour TWA	1992	
†N,N-Dimethyl-formamide [68-12-2]	10 ppm	8- and 12-hour TWA, skin Biological Exposure Index (BEI) = 20 ppm of MMF in an end-of-shift urine sample for several workers doing the same job and 40 ppm for an individual result	1993	(NC 1993)* (D 1993)* (R 1993)* (M 1993)*
†1,1-Dimethyl-hydrazine [57-14-7]	0.01 ppm	8-hour TWA, skin	1991	C-A 1991 (D 1991)* (R 1991)* (M 199[illegible]*
N,O-Dimethyl-hydroxylamine [1117-97-1]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†Dimethylnitrosamine [62-75-9]	0.8 ppb 2.4 ppb	8-hour TWA 15-minute TWA	1992	
†Dimethyl Sulfate [77-78-1]	0.01 ppm	8- and 12-hour TWA, skin	1993	C-A 1992 (D 1992)*
Dimethyl Sulfide [75-18-3]	10 ppm	8-hour TWA Odor may require lower limit	1990	
†Dimethylsulfoxide (DMSO) [67-68-5]	10 ppm	8-hour TWA, skin	1990	d 1991 (NC 1991)* (M 1991)*
Dimethyl Terephthalate [120-61-6]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1988	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†2,4-Dinitrotoluene [121-14-2]	0.15 mg/m³	8- and 12-hour TWA, skin ≤ 5%, 2,6-DNT	1992	C-A 1983
1,4-Dioxane [123-91-1]	25 ppm	8-hour TWA, skin	1990	c 1975
Dioxolane 416 (4,4,5,5-Tetra-chloro-2,2-bis (trifluoromethyl)-1,3-dioxolane [64499-81-6]	0.1 ppm	8-hour TWA	1988	r 1987
Dioxolane 418 (4,5-Dichloro-4,5-difluoro-2,2-bis (trifluoromethyl)-1,3-dioxolane [60644-92-0] [cis-isomer 64499-82-7] [trans-isomer 64499-83-8]	25 ppm	8-hour TWA	1988	
Dioxolane 456 (2,2-bis(Tri-fluoromethyl)-1,3-dioxolane) [1765-26-0]	0.1 ppm	8-hour TWA	1988	r 1987
Dioxole 418 (4,5-Difluoro-2,2-bis-(trifluoro-methyl)-1,3-dioxole [37697-64-6]	25 ppm	8-hour TWA	1988	
†Diuron [330-54-1]	1 mg/m³	8- and 12-hour TWA, total dust	1993	c 1993 (D 1993)* (R 1993)*
†1,12-Dodecanediamine [2783-17-7]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Dodecanedioic Acid [693-23-2]	5 mg/m³	8- and 12-hour TWA, respirable dust	1989	
	10 mg/m³	8- and 12-hour TWA, total dust		
n-Dodecyl Mercaptan [112-55-0]	1 ppm	8-hour TWA	1989	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Dow-Corning 5772 (DC-5772) [27668-52-6]	0.05 mg/m³	8-hour TWA	1992	
†Dowtherm® A [8004-13-5]		Use ACGIH mixture formula and AELs for biphenyl and biphenyl ether	1992	
†DPX-66037 [126535-15-7]	2 mg/m³	8- and 12-hour TWA	1994	
DPX-79376		See Quizalofop Ethyl, D+ Isomer		
DPX-A7881 [97780-06-8]	5 mg/m³	8- and 12-hour TWA, respirable dust	1989	
	10 mg/m³	8- and 12-hour TWA, total dust		
DPX-E9636 (Used in Titus® Herbicide) [122931-48-0]	5 mg/m³	8- and 12-hour TWA, total dust	1992	
DPX-F6025		See Chlorimuron Ethyl		
†DPX-L5300 (Used in Express® Herbicide) [101200-48-0]	1 mg/m³	8-hour TWA	1992	(c 1987)* (D 1987)* (R 1987)*
DPX-M6316 (Used in Harmony® Weed Killer) [79277-27-3]	5 mg/m³	8- and 12-hour TWA	1988	
DPX-V9360		See Nicosulfuron		
DS-19 Silicone Emulsion	0.05 mg/m³	8-hour TWA	1992	
Dytek® A Amine [15520-10-2]	0.4 ppm	8- and 12-hour TWA (vapor)	1991	
	2 mg/m³	8- and 12-hour TWA (particulate)		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Elvacite® (polymethacrylate) [9011-14-7]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Emery 6724 [109909-40-2]	0.1 mg/m³	8-hour TWA	1990	
Epichlorohydrin [106-89-8]	----	OSHA PEL = 2 ppm, skin ACGIH TLV = 2 ppm, skin		c 1977
†1,2-Epoxy-3-phenoxy-propane (EPP, phenyl glycidyl ether) [122-60-1]	0.75 ppm	8-hour TWA	1990	C-A 1979
†Epoxy Resins	----	Maintain Epichlorohydrin TLV of 2 ppm, skin ELC Reclassification Pending		c 1981
Esfenvalerate (Used in Asana® Insecticide) [66230-04-4]	2 mg/m³	8- and 12-hour TWA, skin	1988	
Ethanol [64-17-5]	1000 ppm	8- and 12-hour TWA	1990	
†Ethanolamine [141-43-5]	3 ppm	8- and 12-hour TWA	1992	
†Ethion [563-12-2]	0.4 mg/m³	8-hour TWA, skin	1992	
†2-Ethoxyethanol (Cellosolve®, Dovanol® EE) [110-80-5]	5 ppm	8-hour TWA, skin	1992	(NC 1991)* D 1991 R 1991 (M 1991)*
2-Ethoxyethyl acetate (Cellosolve® acetate) [111-15-9]	5 ppm	8-hour TWA, skin	1992	D 1991 R 1991 (M 1991)*

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Ethyl Acrylate [140-88-5]	2 ppm	8-hour TWA, skin	1990	(c 1990)* (D 1990)*
Ethyl Chloride [75-00-3]	150 ppm	8-hour TWA	1989	c 1989 (D 1989)*
Ethyl Chloroformate [541-41-3]	0.5 ppm 1 ppm	8- and 12-hour TWA 15-minute TWA	1991	
Ethylene Chlorohydrin [107-07-3]	1 ppm	15-minute TWA, skin	1989	
†Ethylene Dibromide [106-93-4]	20 ppb	8- and 12-hour TWA, skin	1991	C-A 1991 R 1991 (D 1991)* (M 1991)*
Ethylene Dichloride [107-06-2]	1 ppm	8- and 12-hour TWA, skin	1991	c 1991 (R 1991)* (D 1991)* (M 1991)*
†Ethylene Glycol [107-21-1]	50 ppm 10 mg/m³	8-hour TWA, vapor 8-hour TWA, particulate	1986	(NC 1985)* (D 1985)* (R 1985)*
Ethylene Glycol Dimethacrylate [97-90-5]	5 ppm	8-hour TWA	1990	
†Ethylene Oxide [75-21-8]	1 ppm	8-hour TWA OSHA PEL = 1 ppm (8-hour TWA)	1986	C-A 1985 (D 1985)* r 1985

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Ethylenethiourea [96-45-7]	0.2 mg/m³	8-hour TWA, skin	1992	C-A 1975 D 1972
		Limit is not for fetal exposure		
	0.2 ppm	Average urinary acceptable level		
	0.4 ppm	Limit for a single urinary sample, requires follow-up action.		
†2-Ethylhexanol [104-76-7]	20 ppm	8-hour TWA	1989	(c 1992)* (D 1992)* (M 1992)*
2-Ethylhexyl Acrylate [103-11-7]	5 ppm	8-hour TWA, skin	1991	c 1986
2-Ethylhexyl Methacrylate [688-84-6]	25 ppm	8-hour TWA	1988	
†2-Ethylhexyl Nitrate [27247-96-7]	5 ppm	8- and 12-hour TWA	1992	
Ethyl Methacrylate [97-63-2]	25 ppm	8-hour TWA	1988	
Express® Herbicide		See DPX-L5300		
Exxate 900 [108419-33-6]	25 ppm	8-hour TWA	1990	
FC-116 (Hexafluoro-ethane) [76-16-4]	1000 ppm	8- and 12-hour TWA	1988	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
FC-C-51-12 (Hexafluoro-1,2-bis-(trifluoromethyl)-cyclobutane [2994-71-0] mixture with hexafluoro-1,3-bis(trifluoromethyl)-cyclobutane [13221-71-1]	1000 ppm	8- and 12-hour TWA	1992	
Fenbutatin Oxide (Used in Vendex® Miticide) [13356-08-6]	0.1 mg/m³	8- and 12-hour TWA, total dust	1989	
†Fenvalerate [51630-58-1]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†Fiber Glass	2 fibers per cc	8-hour TWA Respirable fibers < 3 u in diameter, > 5 u length, and with an aspect ratio > 3:1	1989	(c 1989)*
	5 mg/m³	8-hour TWA, non-fibrous particulate and/or non-respirable fibers		
Fluorobenzene [462-06-6]	25 ppm	8- and 12-hour TWA	1993	
†Flusilazole (INH-6573) [85509-19-9]	0.5 mg/m³	8-hour TWA	1994	c 1994 d 1994 (R 1994)*
Folpet [133-07-3]	5 mg/m³	8-hour TWA	1986	
Formaldehyde [50-00-0]	1.0 ppm 2.0 ppm	8- and 12-hour TWA 15-minute TWA	1987	c 1981
†Formamide [75-12-7]	10 ppm	8-hour TWA, skin	1992	D 1986
†Fortress® Insecticide		See Chlorethoxyfos		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Fullers Earth (Attapulgite Clay) [8031-18-3]	1 mg/m³	8-hour TWA	1990	
Gardona® Insecticide		See Tetrachlorvinphos		
Glean® Herbicide		See Chlorsulfuron		
†Glutaraldehyde [111-30-8]	0.03 ppm 0.1 ppm	8-hour TWA 15-minute TWA	1993	
Glycidyl Methacrylate -[106-91-2]	1 ppm	8-and 12-hour TWA, skin	1990	
†Glycolic Acid [79-14-1]	10 mg/m³	8- and 12-hour TWA	1992	
†Halon 2402 [124-73-2]	100 ppm	8-hour TWA	1992	
Harmony® Weed Killer		See DPX-M6316		
HCFC-21 (Dichloro-fluoromethane) [75-43-4]	10 ppm	8-hour TWA	1990	
†HCFC-22 (Chloro-difluoromethane) [75-45-6]	-----	ACGIH TLV = 1000 ppm	-----	(c 1981)* (D 1978)*
HCFC-31 (Chlorofluoro-methane) [593-70-4]	10 ppm	8- and 12-hour TWA	1992	c 1992 r 1992(males) d 1992
HCFC-122 (1,2,2-Trichloro-1,1-difluoroethane) [354-21-2]	10 ppm	8- and 12-hour TWA	1991	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†HCFC-123 (2,2-Dichloro-1,1,1-trifluoroethane) [306-83-2]	30 ppm	8- and 12-hour TWA	P1994	
HCFC-124 (2-Chloro-1,1,1,2-tetrafluoroethane [2837-89-0]	500 ppm	8- and 12-hour TWA	1989	
HCFC-132b (1,2-Dichloro-1,1-difluoroethane) [1649-08-7]	5 ppm	8- and 12-hour TWA	1989	Interim Guidance Letter 5/29/87
†HCFC-133a (2-Chloro-1,1,1-trifluoroethane) [75-88-7]	5 ppm	8- and 12-hour TWA	1992	c 1987 D 1987 R 1987
HCFC-141b (1,1-Dichloro-1-fluoroethane) [1717-00-6]	500 ppm	8- and 12-hour TWA	1991	
HCFC-142b (1-Chloro-1,1-difluoroethane) [75-68-3]	1000 ppm	8-hour TWA	1990	
Hexachloroacetone [116-16-5]	0.2 ppm	8-hour TWA	1988	
1,4-Hexadiene [592-45-0]	10 ppm	8- and 12-hour TWA	1991	
Hexafluoroacetone [684-16-2]	0.1 ppm	8- and 12-hour TWA, skin; Limit is for men, and women not of childbearing capability	1989	D 1989 R 1989
	0.005 ppm	8- and 12-hour TWA, skin; Limit is for women of childbearing capability; Skin contact must be entirely avoided		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Hexafluoro-1,2-bis-(trifluoromethyl)-cyclobutane mixture with hexafluoro-1,3-bis-(trifluoromethyl)cyclobutane		See FC-C-51-12		
Hexafluoroethane		See FC-116		
†Hexafluoro-isopropanol [920-66-1]	10 ppm	8-hour TWA	1992	
Hexafluoropropylene [116-15-4]	2 ppm	8-hour TWA	1992	
Hexafluoropropylene Epoxide [428-59-1]	20 ppm	8-hour TWA	1990	
Hexamethylenediamine [124-09-4]	1 ppm	8- and 12-hour TWA, vapor	1989	
	5 mg/m³	8- and 12-hour TWA, total particulate		
Hexamethyleneimine [111-49-9]	0.5 ppm	8- and 12-hour TWA	1989	
†Hexamethylphosphor-amide (HMPA) [680-31-9]	0.5 ppb	8-hour TWA	1990	C-A 1975
n-Hexane [110-54-3]	50 ppm	8- and 12-hour TWA	1993	
†Hexazinone (Used in Velpar® Herbicide) [51235-04-2]	10 mg/m³	8-hour TWA	1992	(c 1993)* (R 1993)* (D 1993)*

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Hexythiazox (Used in Savey® Miticide [78587-05-0]	2 mg/m³	8-hour TWA	1992	c 1987 (D 1987)* (R 1987)*
HFC-23 (Trifluoro-methane) [75-46-7]	1000 ppm	8- and 12-hour TWA	1988	
HFC-32 (Difluoro-(methane) [75-10-5]	1000 ppm	8- and 12-hour TWA	1991	
HFC-125 (Penta-fluoroethane) [354-33-6]	1000 ppm	8- and 12-hour TWA	1988	
HFC-134 (1,1,2,2-Tetra-fluoroethane) [359-35-3]	1000 ppm	8- and 12-hour TWA	1993	
†HFC-134a (1,1,1,2-Tetra-fluoroethane) [811-97-2]	1000 ppm	8- and 12-hour TWA	1994	
HFC-143a (1,1,1-Trifluoro-ethane) [420-46-2]	1000 ppm	8- and 12-hour TWA	1992	
HFC-152a (1,1-Difluoro-ethane) [75-37-6]	1000 ppm	8-hour TWA	1990	
HFC-338pcc (1,1,2,2,3,3,4,4-octafluorobutane) [377-36-6]	500 ppm	8- and 12-hour TWA	1993	
†HFC-43-10 (1,1,1,2,2,3,4,5,5,5-Decafluoropentane) [138495-42-8]	400 ppm	8- and 12-hour TWA	1992	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
HFPO Dimer [2062-98-8]	1 ppm	8- and 12-hour TWA	1991	
†Hydrazine [302-01-2]	0.01 ppm	8- and 12-hour TWA, skin	1992	C-A 1991 (D 1991)* (M 1991)*
†Hydrazine Sulfate [10034-93-2]	0.01 ppm	8- and 12-hour TWA, skin	1992	C-A 1991 (D 1991)* (M 1991)*
†Hydrazoic Acid [7782-79-8]	0.05 ppm	8-hour TWA ELC Reclassification	1992 Pending	(C 1985)* (D 1985)*
Hydrogen Chloride [7647-01-0]	5 ppm	15-minute TWA	1990	
Hydrogen Cyanide [74-90-8]	10 ppm 5 ppm	8-hour TWA, skin 12-hour TWA, skin	1990	
Hydrogen Fluoride [7664-39-3]	3 ppm	15-minute TWA	1991	
†Hydrogen Sulfide [7783-06-4]	10 ppm	8- and 12-hour TWA	1992	
Hydroquinone [123-31-9]	2 mg/m³	8- and 12-hour TWA	1992	c 1992 (R 1992)* (D 1992)*
Hydroxyethyl Acrylate [818-61-1]	1 ppm 3 ppm	8- and 12-hour TWA, skin 15-minute TWA, skin	1988	
†N-(2-Hydroxyethyl) ethyleneimine [1072-52-2]	----	ELC Reclassification	Pending	C 1976
2-Hydroxyethyl methacrylate [868-77-9]	25 ppm	8-hour TWA	1988	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Hytrel® Polyester Elastomer [60130-75-8]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1990	
†IN-43898		See Chlorethoxyfos		
†IN-70247 (Methyl 2-(chlorosulfonyl)-3-methylbenzoate [126535-26-0]	1 mg/m³	8-hour TWA	P1994	
†INB-4450 (Ethyl 2-(aminosulfonyl)-benzoate) [59777-72-9]	10 mg/m³	8- and 12-hour TWA, total dust	1992	
IND-5803 (2-Carbomethoxybenzenesulfonamide) [57683-71-3]	10 mg/m³	8- and 12-hour TWA, total dust	1990	
IND-7556 (6-Ethoxy-N-methyl-1,3,5-triazine-2,4-diamine [62096-63-3]	1 mg/m³	8- and 12-hour TWA, total dust	1991	
†IND-8526 (N2,N2-Dimethyl-6-(2,2,2-trifluoroethoxy)-1,3,5-triazine-2,4-diamine) [145963-84-4]	1 mg/m³	8-hour TWA	P1994	
INF-5384		See Bensulfuron Methyl		
INH-1043 (2-Amino-6-hydroxy-4(1H)-pyrimidinone) [56-09-7]	10 mg/m³	8-hour TWA, total dust	1991	
†INH-1044 (4,6-Dichloro-2-pyrimidinamine) [56-05-3]	10 mg/m³	8-hour TWA, total dust	1993	
INH-6573		See Flusilazole		

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†INJ-290 (4,6-Dimethoxy-2-pyrimidinamine) [36315-01-2]	10 mg/m³	8-hour TWA, total dust	1993	
†INL-5296 (N,6-Dimethyl-4-methoxy-1,3,5-triazine-2-amine) [5248-39-5]	1 mg/m³	8- and 12-hour TWA	1992	
INL-5300		See DPX-L5300		
INM-6316		See DPX-M6316		
INN-5297 (Methyl 2-[(amino-sulfonyl)methyl]benzoate)	10 mg/m³	8- and 12-hour TWA, total dust	1989	
INN-6186 (4-Chloro-6-methoxy-2-pyrimidinamine [5734-64-5]	10 mg/m³	8-hour TWA, total dust	1991	
INT-6376		See Metsulfuron Methyl		
INU-9069 [112006-94-7]	10 mg/m³	8- and 12-hour TWA, total dust	1992	
INV-9367 [112006-75-4]	10 mg/m³	8- and 12-hour TWA, total dust	1992	
†INX-993 (4,6-Dimethyl-2-pyrimidinamine) [767-15-7]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1992	
INY-5893		See Hexythiazox		
Irganox® 245 [36443-68-2]	1 mg/m³	8-hour TWA	1989	c 1989 (D 1989)*
Isoheptane [31394-54-4]	300 ppm	8-hour TWA Hydrocarbon mixture, See documentation for composition	1990	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Isopar® E [64742-48-9]	100 ppm	8- and 12-hour TWA	1994	
Isopar® G [64742-48-9]	100 ppm	8-hour TWA	1988	
Isopar® L [64742-48-9]	100 ppm	8-hour TWA	1988	
†Isophthalic Acid [121-91-5]	10 mg/m³	8-hour TWA, total dust	1992	
	5 mg/m³	8-hour TWA, respirable dust		
†Isophthaloyl Chloride [99-63-8]	0.5 ppm	8- and 12-hour TWA	1993	
	1.0 ppm	15-minute TWA		
Isopropyl Alcohol [67-63-0]	400 ppm	8- and 12-hour TWA	P1991	
Kapton® (polyimide) [25038-81-7]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Kathon® CG/ICP		See 5-Chloro-2-methyl-3(2H)-isothiazolone mixture with 2-methyl-3(2H)-isothiazolone		
Kelthane (1,1-Bis-p-chlorophenyl)-2,2,2-trichloroethanol) [115-32-2]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†Kevlar® (fibrils) [24938-64-5]	2 fibrils per cc	8-hour TWA, Respirable fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio > 3:1	1993	(NC 1993)*
	5 mg/m³	8-hour TWA for non-fibrous particulate and/or non-respirable fibers		

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Lactic Acid [50-21-5]	5 mg/m³	8- and 12-hour TWA	1993	
Lead and inorganic and organic lead compounds		See Hazard Determination Letter dated July 10, 1989 for information on recommended blood lead levels; OSHA PEL for inorganic lead compounds, including lead soaps is 0.05 mg/m³; OSHA PEL for TEL and TML is 0.075 mg/m³, skin	1989	D 1989 r 1989
†Lead Chromate [7758-97-6]	0.05 mg/m³	8-hour TWA, as chromium; Also see Lead Hazard Determination Letter dated July 10, 1989	1992	C-H 1975 D 1989 r 1989
Lead Naphthenate [61790-14-5]	0.05 mg/m³	8-hour TWA, skin; as lead. Also see the Lead Hazard Determination Letter dated July 10, 1989	1989	c 1989 D 1989 r 1989
†Lenacil Herbicide [2164-08-1]	5 mg/m³	8- and 12-hour TWA, total dust	1994	(c 1993)* (D 1993)* (R 1993)*
Leucopure® EGM [3333-62-8]	10 mg/m³	8-hour TWA	1990	
Light Green SF [5141-20-8]	----	ELC Reclassification Pending		(C 1980)*
†d-Limonene [5989-27-5]	50 ppm	8-hour TWA	1993	(c 1989)* (D 1989)*
Lindane [58-89-9]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Linuron [330-55-2]	2 mg/m³	8- and 12-hour TWA, total dust	1990	c 1983

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Lithium Compounds which include: LiCl [7447-41-8] LiBr [7550-35-8] LiI [10377-51-2] Li_2CO_3 [554-13-2] Li Glycolate [23248-23-9] Lithium Sulfate [10377-48-7]	1 mg/m³	8-hour TWA, as lithium	1992	d 1982
Londax® Herbicide		See Bensulfuron Methyl		
†Lontrel® (3,6-Dichloro-2-pyridinecarboxylic acid) [1702-17-6]	10 mg/m³	8-hour TWA	1992	
Lucite® (polymethacrylate) [9011-14-7]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Ludox® Colloidal Silica	1 mg/m³	8-hour TWA, as respirable silica dust	1990	
Maleic Anhydride [108-31-6]	0.1 ppm	8- and 12-hour TWA	1991	
†Maleic Hydrazide [123-33-1]	10 mg/m³	8-hour TWA	1992	
Mancozeb (Used in Manzate® Fungicide) [8018-01-7]	2.0 mg/m³	8-hour TWA, total dust	1990	c 1989 (D 1989)* (R 1989)*
	1.5 mg/m³	12-hour TWA, total dust		
Maneb [12427-38-2]	2 mg/m³	8-hour TWA, total dust	1988	
	1.5 mg/m³	12-hour TWA, total dust		
Manzate® Fungicide		See Mancozeb		

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
MCPA (4-Chloro-2-methyl-phenoxyacetic acid) [94-74-6]	2 mg/m³	8-hour TWA	1986	
Mecoprop (2-(4-chloro-2-methylphen-oxy)propanoic acid) [93-65-2]	2 mg/m³	8-hour TWA	1991	
Methacrylic Acid [79-41-4]	10 ppm	8-hour TWA	1988	
†Methacrylonitrile [126-98-7]	1 ppm	8- and 12-hour TWA, skin	1992	
†Methanol [67-56-1]	200 ppm	8- and 12-hour TWA, skin	1994	d 1994
†2-Methoxyethanol (Methyl Cellosolve® Dowanol® EM) [109-86-4]	1 ppm	8-hour TWA, skin	1992	D 1982 R 1982
†2-Methoxyethyl acetate (Methyl Cellosolve® acetate) [110-49-6]	1 ppm	8-hour TWA, skin	1992	D 1986 R 1986
Methoxyisobutyl-isonitrile (MIBI)	50 ppb	8-hour TWA	1988	
p-Methoxyphenol (MEHQ) [150-76-5]	2 mg/m³	8-hour TWA	P1992	
Methyl Acetoacetate [105-45-3]	200 ppm	8-hour TWA	1990	
Methyl Acrylate [96-33-3]	2 ppm	8- and 12-hour TWA, skin	1993	
†Methylamine [74-89-5]	10 ppm	8- and 12-hour TWA	1992	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
MBC (Methyl 2-Benzimidazolecarbamate) [10605-21-7]	5 mg/m³	8- and 12-hour TWA, total dust	1991	c 1991 r 1991(males) (D 1991)* (M 1991)*
2-Methyl-1,4-butanediol [2938-98-9]	30 ppm	8- and 12-hour TWA	1991	
Methyl Cellosolve®		See 2-Methoxyethanol		
Methyl Cellosolve® Acetate		See 2-Methoxyethyl Acetate		
Methyl Chloride [74-87-3]	50 ppm 100 ppm	8-hour TWA 15-minute TWA	1990	c 1985 (D 1985)* r 1985
Methyl Chloroformate [79-22-1]	0.5 ppm 1 ppm	8- and 12-hour TWA 15-minute TWA	1991	
Methyl 2,2-difluoromalonyl fluoride [69116-71-8]	0.1 ppm	8- and 12-hour TWA	1991	
†4,4'-Methylenebis-(2-chloroaniline) (MOCA®) [101-14-4]	----	ACGIH TLV = 20 ppb; A2 carcinogen		C-A 1978
Methylenebis-(4-phenyl-isocyanate) (MDI) [101-68-8]	0.02 ppm	20-minute TWA	1986	
†Methylene Chloride [75-09-2]	50 ppm 25 ppm	8-hour TWA 12-hour TWA	1993	c 1993 (D 1993)* (R 1993)*
†4,4'-Methylenedianiline [101-77-9]	0.1 mg/m³	8- and 12-hour TWA, skin	1984	C-A 1983
†4,4'-Methylenedianiline dihydrochloride [13552-44-8]	0.1 mg/m³	8- and 12-hour TWA, skin	1990	C-A 1983

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Methyl Ethyl Ketone [78-93-3]	200 ppm 300 ppm	8- and 12-hour TWA 15-minute TWA	1989	
Methyl Ethyl Ketoxime [96-29-7]	50 ppm	8-hour TWA	1990	
2-Methylglutaronitrile [4553-62-2]	1 ppm	8-hour TWA, skin	P1989	
N-Methylolacrylamide [924-42-5]	0.25 ppm	8-hour TWA	1989	c 1989 r 1989(males)
3-Methylpiperidine [626-56-2]	1 ppm	8- and 12-hour TWA, skin	1992	
†N-Methyl-2-pyrrolidinone [872-50-4]	25 ppm	8-hour TWA	1989	(NC 1988)* (D 1988)* (R 1988)*
3-Methyltetrahydrofuran [13423-15-9]	200 ppm	8- and 12-hour TWA	1991	
Methyl p-Toluate [99-75-2]	20 ppm	8-hour TWA	1990	
Methyltrichlorosilane [75-79-6]	5 ppm	15-minute TWA, as HCl	1989	
Metribuzin [21087-64-9]	5 mg/m³	8-hour TWA	1990	
Metsulfuron Methyl (Used in Ally® Weed Killer) (INT-6376) [74223-64-6]	10 mg/m³	8- and 12-hour TWA	1990	
†Microwave Radiation		See ACGIH TLV for guidance		

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†Mineral Wool	1 fiber/cc	8-hour TWA Respirable fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio > 3:1	1989	(c 1989)*
	5 mg/m³	8-hour TWA, non-fibrous particulate and/or non-respirable fibers		
Monomethylformamide [123-39-7]	2 ppm	8- and 12-hour TWA, skin	1988	(D 1989)*
Monsanto Benzyl Chloride Residue		See Benzyl chloride Residue (BCR)		
†NER-010A Epoxy Resin [25038-04-4]	0.1 mg/m³	8- and 12-hour TWA Maintain ACGIH TLV of 2 ppm, skin for epichlorohydrin	1993	c 1993
†Nickel & Inorganic Nickel Compounds [7440-02-0 Nickel Metal] [557-19-7 Nickel Cyanide]	0.02 mg/m³	8- and 12-hour TWA, as nickel	1991	C-H 1991 r 1991 (D 1991)* (M 1991)*
Nickel Tritolyl-phosphite [35884-66-3]	0.1 mg/m³ 0.05 mg/m³	8-hour TWA 12-hour TWA	1985	
Nicosulfuron (Used in Accent® Herbicide) [111991-09-4]	5 mg/m³	8- and 12-hour TWA, respirable dust	1990	
†Nitric Acid [7697-37-2]	5 mg/m³	8- and 12-hour TWA	1992	
o-Nitroaniline [88-74-4]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
p-Nitroaniline [100-01-6]		Not currently in use within DuPont. See the List of Inactive AELs for details.		

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†5-Nitro-o-anisidine [99-59-2]	0.5 mg/m³	8-hour TWA, skin	1992	c 1980
†Nitrobenzene [98-95-3]	0.1 ppm	8- and 12-hour TWA, skin	1993	C-A 1993 r 1993 (D 1993)*
†p-Nitrobenzoic Acid [62-23-7]	2 mg/m³	8- and 12-hour TWA, skin	1994	c 1993 (R 1993)*
p-Nitrobenzyl Chloride [100-14-1]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
m-Nitrochloro-benzene [121-73-3]	3 ppm	Not currently in use within DuPont. See the List of Inactive AELs for details.		
o-Nitrochloro-benzene [88-73-3]	3 ppm 1 mg/m³	8- and 12-hour TWA (vapor) and 8- and 12-hour TWA (particulate)	1992	
p-Nitrochloro-benzene [100-00-5]	0.1 ppm	8-hour TWA, skin	1990	
Nitrogen Dioxide [10102-44-0]	3 ppm	8- and 12-hour TWA	1989	
†2-Nitronaphthalene [581-89-5]	----	ELC Reclassification Pending		C 1976
N-Nitrosodiphenyl-amine [86-30-6]	----			c 1980
Nitrosylsulfuric Acid [7782-78-7]	1 mg/m³	8-hour TWA	1990	
†o-Nitrotoluene [88-72-2]	1 ppm	8- and 12-hour TWA	1992	C-A 1992

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†Nitrous Oxide [10024-97-2]	50 ppm	8- and 12-hour TWA	1992	(NC 1991)* R 1991 D 1991
Nomex® (polyamide) [25765-47-3]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Norbornadiene [121-46-0]	100 ppm	8- and 12-hour TWA	1990	
†Oil Mist	5 mg/m³	8- and 12-hour TWA	1992	
Optisol In-Line Solution (Mixture of C6-C8 Branched Chain Alkyl Alcohols and C6-C8 Branched Chain Alkyl Acetic Acid Esters)	0.5 ppm	8-hour TWA, alcohol	1993	
	5 ppm	15-minute TWA, alcohol and		
	1 ppm	8-hour TWA, ester		
	10 ppm	15-minute TWA, ester		
†Orlon® (polyacrylonitrile) [9010-78-0 and 25014-41-9]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Oust® Herbicide		See Sulfometuron Methyl		
†Oxalic Acid [144-62-7]	1 mg/m³	8- and 12-hour TWA	1994	
Oxamyl [23135-22-0]	0.5 mg/m³	8-hour TWA	1990	
	1.0 mg/m³	15-minute TWA		
Oxone® [70693-62-8]	1 mg/m³	8-hour TWA	1988	
†Oxydianiline [101-80-4]	0.1 mg/m³	8-hour TWA	1992	C-A 1987
	0.3 mg/m³	15-minute TWA		(R 1987)*
†Pentaerythritol Triacrylate [3524-68-3]	----	WEEL = 1 mg/m³		(NC 1988)* (D 1988)*

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Pentafluoroallyl chloride [79-47-0]	0.2 ppm 0.5 ppm	8-hour TWA 15-minute TWA	1988	
Pentafluoroethane		See HFC-125		
†Pentafluoropropionyl peroxide (3P) [356-45-6]	15 ppb	8-hour TWA	1992	
†n-Pentane [109-66-0]	600 ppm	8- and 12-hour TWA	1993	
2,4-Pentanedione [123-54-6]	10 ppm	8-hour TWA	1990	(D 1990)*
†2-Pentenenitrile		See Stripped 2-Pentenenitrile		
3-Pentenenitrile [4635-87-4]	5 ppm 3 ppm	8-hour TWA, skin 12-hour TWA, skin	1988	
Perfluorobutyl Iodide [423-39-2]	50 ppm	8-hour TWA	1991	
Perfluorobutylethyl Iodide [2043-55-2]	5 ppm	8-hour TWA	1990	
†Perfluorobutyl-ethylene [19430-93-4]	100 ppm	8-hour TWA	1992	
Perfluoroiso-butylene (PFIB) [382-21-8]	0.01 ppm 0.03 ppm	8-hour TWA 15-minute TWA	1990	
†Peroxyacetic Acid [79-21-0]	0.2 ppm	8- and 12-hour TWA	1992	
Petro® AG [26264-58-4]	10 mg/m³	8-hour TWA	1990	

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
2-Phenyl-APB (1,4-bis(4-Amino-phenoxy)-2-phenylbenzene [94148-67-1]	0.1 mg/m³	8-hour TWA	1992	
Phenyl Chloroformate [1885-14-9]	0.5 ppm 1 ppm	8- and 12-hour TWA 15-minute TWA	1991	
†m-Phenylene-diamine [108-45-2]	0.1 mg/m³	8- and 12-hour TWA	1994	
o-Phenylene-diamine [95-54-5]	0.1 mg/m³	8- and 12-hour TWA, skin	1991	c 1991 (M 1991)*
†p-Phenylene-diamine [106-50-3]	0.1 mg/m³	8- and 12-hour TWA	1994	
†p-Phenylene-diisocyanate (PPDI) [104-49-4]	0.03 mg/m³	8- and 12-hour TWA, particulate and vapor combined	1993	
Phenyl glycidyl ether		See 1,2-Epoxy-3-phenoxypropane		
Phenylhydroquinone [1079-21-6]	2 mg/m³	8-hour TWA	1990	
o-Phenylphenol [90-43-7]	1 mg/m³	8-hour TWA	1990	
Phosgene [75-44-5]	0.1 ppm	15-minute TWA	1989	
Phosphoric Acid [7664-38-2]	1 mg/m³	8- and 12-hour TWA	1990	
Phosphorous Trichloride [7719-12-2]	3 mg/m³	8- and 12-hour TWA	1990	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
beta-Picoline [108-99-6]	2 ppm	8-hour TWA, skin	1990	
Polyethylene Glycol 400 [9081-95-2]	10 mg/m³	8-hour TWA, total particulate	1991	
	5 mg/m³	8-hour TWA, respirable particulate		
Polyethylene Terephthalate (Dacron®, Mylar®) [9003-68-3 and 25038-59-9]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Poly-Fill® 80C [1344-95-2]	5 mg/m³	8-hour TWA	1990	
Polyvinyl Alcohol [9002-89-5]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Potassium Cyanide [151-50-8]	5 mg/m³	8-hour TWA, as cyanide	1990	
Potassium Iodide [7681-11-0]	10 mg/m³	8-hour TWA	1992	
Potassium Sulfate [7778-80-5]	10 mg/m³	8-hour TWA	1990	
Potassium Tripolyphosphate [13845-36-8]	10 mg/m³	8-hour TWA	1990	
1-Propanol [71-23-8]	200 ppm	8-hour TWA	1989	Carcinogen study not adequate for classification (D 1988)* (R 1988)*

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Propazine [139-40-2]	0.5 mg/m³	8- and 12-hour TWA	1988	c 1987 (D 1987)*
beta-Propiolactone [57-57-8]	----	ACGIH TLV = 0.5 ppm; A2 carcinogen, OSHA regulated		c 1975
†Propylene Glycol Monomethyl Ether Acetate [108-65-6]	10 ppm	8- and 12-hour TWA	1992	(D 1993)*
†Propyleneimine [75-55-8]	----	OSHA = 2 ppm, skin ACGIH TLV = 2 ppm, skin; A2 carcinogen ELC Reclassification Pending		C 1976
†Propylthiouracil [51-52-5]	----			c 1980
Pyromellitic Acid [89-05-4]	0.5 mg/m³	8-hour TWA	1988	
Pyromellitic Dianhydride [89-32-7]	0.5 mg/m³	8-hour TWA	1988	
Quilon® Chrome Complex [15242-96-3]	0.5 mg/m³	8-hour TWA, as chromium	1991	
Quinacridone [1047-16-1]	10 mg/m³	8-hour TWA	1990	
Quizalofop Ethyl (D+ Isomer Used in Assure® Herbicide) [100646-51-3]	2 mg/m³	8-hour TWA, total dust	1990	c 1991 (D 1991)* (R 1991)* (M 1991)*
Rabon® Insecticide		See Tetrachlorvinphos		

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June 17, 1994

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Refractory Aluminum Silicate Ceramic Fibers [142844-00-6]	0.2 fibers/cc	8- and 12-hour TWA Respirable fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio of > 3:1 See documentation for special handling procedures	1993	C-A 1993
	5 mg/m³	8-hour TWA for non-fibrous particulate and/or non-respirable fibers		
Resorcinol Oxydianiline (RODA) [2479-46-1]	0.5 mg/m³	8- and 12-hour TWA	1991	
Savey® Miticide		See Hexythiazox		
†Saytex® 120 [58965-66-5]	5 mg/m³	8-hour TWA, total dust	1992	
†Siduron [1982-49-6]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Silica, Amorphous (Cab-O-Sil®, Syloid®) [7631-86-9]	6 mg/m³	8-hour TWA, total dust	1988	
	3 mg/m³	8-hour TWA, respirable dust		
Silica, Crystalline (Quartz) [14808-60-7]	0.1 mg/m³	8-hour TWA, respirable dust	1988	c 1988
†Silicon Carbide Fibers	0.2 fibers per cc	8-hour TWA Respirable fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio > 3:1	1989	C-A 1989
	5 mg/m³	8-hour TWA for non-fibrous particulate and/or non-respirable fibers		
Sodium Acetate [127-09-3]	10 mg/m³	8-hour TWA	1990	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Sodium Azide [26628-22-8]	0.15 mg/m³	8-hour TWA	1992	(NC 1985)* (D 1985)*
†Sodium Bicarbonate [144-55-8]	10 mg/m³	8-hour TWA, total dust	1992	
	5 mg/m³	8-hour TWA, respirable dust		
†Sodium Bisulfate [7681-38-1]	1 mg/m³	8- and 12-hour TWA	1992	
†Sodium Carbonate [497-19-8]	5 mg/m³	8-hour TWA	1992	
†Sodium Dichromate [10588-01-9]	0.01 mg/m³	8- and 12-hour TWA, as chromium ELC Reclassification Pending	P1994	c 1986
Sodium 1,3-Dimethyl-5-sulfoisophthalate [3965-55-7]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Sodium Gluconate [527-07-1]	10 mg/m³	8-hour TWA	1990	
Sodium Hydroxide [1310-73-2]	2 mg/m³	15-minute TWA	1990	
Sodium Nitrite [7632-00-0]	2 mg/m³	8-hour TWA, respirable dust	P1990	
Sodium p-Nitrophenolate [824-78-2]	10 mg/m³	8-hour TWA	1990	
Sodium Perborate Tetrahydrate [10486-00-7]	5 mg/m³	8-hour TWA, respirable dust	1988	
Sodium Saccharin [128-44-9]	10 mg/m³	8-hour TWA	1988	

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Sodium Styrene Sulfonate [2695-37-6]	10 mg/m³	8-hour TWA	1990	
†Stripped 2-Pentene-nitrile (SPN)	0.3 ppm	8- and 12-hour TWA, skin	1994	
†Strontium Nitrate [10042-76-9]	5 mg/m³	8-hour TWA, total dust	1992	
	2.5 mg/m³	8-hour TWA, respirable dust		
†Styrene [100-42-5]	50 ppm	8- and 12-hour TWA	1989	(NC 1989)*
	100 ppm	15-minute TWA		(D 1989)*
				(R 1989)*
†Sulfamic Acid [5329-14-6]	1 mg/m³	8- and 12-hour TWA	1992	
Sulfometuron Methyl (Used in Oust® Herbicide) (DPX-5648) [74222-97-2]	10 mg/m³	8- and 12-hour TWA	1990	
†Sulfur Dioxide [7446-09-5]	2 ppm	8-hour TWA	1992	
	5 ppm	15-minute TWA		
Sulfuric Acid [7664-93-9]	1 mg/m³	8- and 12-hour TWA	1987	(D 1992)* Data are not sufficient for carcinogenic classification
Sulfuryl Chloride [7791-25-5]	0.2 ppm	8- and 12-hour TWA	1990	
	1 ppm	15-minute TWA		
Surlyn® (ionic resin) [25608-26-8]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Syloid® Amorphous Silica		See Silica, Amorphous		

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Talc (non-asbestiform) [14807-96-6]	2 mg/m³	8- and 12-hour TWA, respirable dust	1988	
Teflon® PEP (TFE-HFP copolymer) [25067-11-2]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Teflon® PFA [26655-00-5]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Teflon® TFE (poly-tetrafluoroethylene) [9002-84-0]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Tefzel® (TFE-ethylene-perfluorobutyl-ethylene terpolymer) [51023-51-9]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
Telomer B Carbamate	1 mg/m³	8-hour TWA, respirable particulate	1990	
Telomer B Citrate Urethane (TBCU) [65530-58-7]	0.1 mg/m³	8- and 12-hour TWA, respirable dust	1990	
Telomer B Methacrylate		See Zonyl® TM		
Telomeric Acid Fluoride	0.2 ppb	8- and 12-hour TWA	1990	
Terbacil Herbicide [5902-51-2]	10 mg/m³	8- and 12-hour TWA	1990	

AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Terephthalic Acid [100-21-0]	10 mg/m³	8-hour TWA, total dust	1988	(c 1988)* (R 1988)*
	5 mg/m³	8-hour TWA, respirable dust		
†Terephthaloyl Chloride [100-20-9]	0.5 ppm 1.0 ppm	8- and 12-hour TWA 15-minute TWA	1993	
Tetrachloroethylene [127-18-4]	25 ppm	8- and 12-hour TWA, skin	1992	c 1986 (D 1986)* (R 1986)*
†Tetrachlorvinphos (Used in Rabon® and Gardona® Insecticides) [22248-79-9]	5 mg/m³	8- and 12-hour TWA	1989	(c 1988)* (R 1988)*
Tetradifon (4-Chlorophenyl-2,4,5-trichlorophenylsulfone) [116-29-0]	2 mg/m³	8-hour TWA	1990	
†Tetraethylene Glycol Diacrylate [17831-71-9]	0.5 mg/m³	8- and 12-hour TWA ELC Reclassification Pending	1988	c 1987 (D 1987)*
1,1,1,2-Tetrafluoro-ethane		See HFC-134a		
Tetrafluoroethylene [116-14-3]	50 ppm	8- and 12-hour TWA	1988	
Tetrahydrofuran [109-99-9]	200 ppm	8- and 12-hour TWA	1991	
Tetrahydrofurfuryl Alcohol [97-99-4]	10 ppm	8- and 12-hour TWA	1992	r 1991

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Tetraisopropyl Titanate [546-68-9]	10 mg/m³	8-hour TWA, total dust	1992	
Tetramethylthiourea [2782-91-4]	----			c 1976 (D 1971)*
Tetramethylurea [632-22-4]	----			d 1967
m-Tetramethyl-xylenediisocyanate [85902-02-9]	0.02 ppm	20-minute TWA	1991	
p-Tetramethyl-xylenediisocyanate [25131-06-0]	0.02 ppm	20-minute TWA	1991	
Thioacetamide [62-55-5]	----			c 1980
†Thiourea [62-56-6]	2 mg/m³	8-hour TWA, skin total dust	1988	C-A 1977
Thiram [137-26-8]	5 mg/m³	8-hour TWA	1990	
†Titanium Dioxide [13463-67-7]	10 mg/m³	8-hour TWA, total dust	1990	(c 1983)*
	5 mg/m³	8-hour TWA, respirable dust		
†Titanium Tetrachloride [7550-45-0]	0.5 mg/m³	8- and 12-hour TWA	1993	(NC 1994)*
†Toluene [108-88-3]	50 ppm	8- and 12-hour TWA	P1994	(NC 1994)* (R 1994)* (D 1994)*

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
2,4-Toluenediamine [95-80-7]		Not currently in use within DuPont. See the List of Inactive AELs for details.		
†Toluene Diisocyanate (TDI) (A mixture of 80% 2,4-isomer [584-84-9] and 20% 2,6-isomer [91-08-7]	0.005 ppm 0.02 ppm	8-hour TWA 15-minute TWA	1987	(c 1987)*
†o-Toluidine [95-53-4]	5 ppm	8-hour TWA, skin	1990	C-A 1990
†o-Toluidine Hydrochloride [636-21-5]	5 ppm	8-hour TWA, skin	1990	C-A 1990
Tricalcium Phosphate [7758-87-4]	10 mg/m³	8-hour TWA	1990	
†1,2,4-Trichloro-benzene [120-82-1]	5 ppm	8-hour TWA	1992	
†2,3,4-Trichloro-butene-1 [2431-50-7]	0.025 ppm 0.10 ppm	8- and 12-hour TWA 15-minute TWA	1991	C-A 1987 (D 1987)* (R 1987)*
1,2,2-Trichloro-1,1-difluoroethane		See HCFC-122		
Trichloroethylene [79-01-6]	50 ppm 200 ppm	8-hour TWA 15-minute TWA	1990	c 1989 (D 1989)* (R 1989)*
1,1,1-Trichloro-2,2,2-trifluoroethane		See CFC-113a		
†Triethanolamine [102-71-6]	3 ppm	8- and 12-hour TWA	P1990	(NC 1989)*
Triethylene Glycol [112-27-6]	2 ppm	8-hour TWA	1990	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Triethylene Glycol Di-2-ethylbutyrate [95-08-9]	10 mg/m³	8-hour TWA	1992	
†Triethylenetetramine [112-24-3]	1 ppm	8-hour TWA, skin	1992	(D 1987)*
Trifluoroacetic acid [76-05-1]	2 ppm	8-hour TWA	1991	
Trifluoroethanol [75-89-8]	1 ppm	8-hour TWA, skin	1991	R 1986(males)
Trifluoromethane		See HFC-23		
Trimellitic Anhydride [552-30-7]	0.05 mg/m³	8-hour TWA	1986	
Trimethylamine [75-50-3]	5 ppm	8- and 12-hour TWA	1990	
Trimethylolpropane triacrylate [15625-89-5]	0.5 mg/m³ 2.0 mg/m³	8-hour TWA 15-minute TWA	1990	
†Trimethyl Phosphate [512-56-1]	0.5 ppm	8-hour TWA	1992	c 1993 R 1993 m 1993
Tritolylphosphite [25586-42-9]	10 ppm 5 ppm	8-hour TWA 12-hour TWA	1985	
Typar® (polypropylene) [9003-07-0]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1988	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Tyvek® (polyethylene) [9002-88-4]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		
†Ucon® 50-HB-660 [9038-95-3]	0.5 mg/m³	8- and 12-hour TWA	1993	
†Ucon® 50-HB-5100 [9038-95-3]	0.01 mg/m³	8- and 12-hour TWA	1993	
Vanadium Pentoxide [1314-62-1]	0.05 mg/m³	8- and 12-hour TWA	1988	
Varsol 1 [8032-32-4]	100 ppm	8-hour TWA	1988	
Vazo® 64 [78-67-1]	1 mg/m³	8-hour TWA	1988	
	0.7 mg/m³	12-hour TWA		
Vazo® 67 [13472-08-7]	1 mg/m³	8-hour TWA	1990	
	0.7 mg/m³	12-hour TWA		
Velpar® Herbicide		See Hexazinone		
Vendex® Miticide		See Fenbutatin Oxide		
Vespel® (polyimide) [25038-81-7]	10 mg/m³	8-hour TWA, total dust	1990	
	5 mg/m³	8-hour TWA, respirable dust		
Vinyl Acetate [108-05-4]	10 ppm	8- and 12-hour TWA	1988	c 1987 (D 1987)* (R 1987)*
4-Vinylcyclohexene [100-40-3]	0.2 ppm	8- and 12-hour TWA	1989	c 1989
†Vinyl Fluoride [75-02-5]	1 ppm	8-hour TWA	1993	C-A 1992

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AEL LIST

CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
†Vinylidene Chloride [75-35-4]	5 ppm 10 ppm	8-hour TWA 15-minute TWA	1987	(c 1986)* (D 1986)* (R 1986)*
†Vinylidene Fluoride [75-38-7]	100 ppm	8-hour TWA	1988	(NC 1987)* (D 1987)* (R 1987)*
Volan® Chrome Complex [15096-41-0]	0.5 mg/m³	8-hour TWA, as chromium	1991	
Wollastonite [13983-17-0]	2 fibers per cc	8-hour TWA Fibers < 3 u in diameter, > 5 u in length, and with an aspect ratio > 3:1	1989	
	5 mg/m³	8-hour TWA for non-fibrous particulate and/or non-respirable fibers		
Wood Dust (Hard and soft wood)	1 mg/m³	8-hour TWA	1990	
†Xylene [1330-20-7] and the o-Xylene Isomer [95-47-6], m-Xylene Isomer [108-38-3], and the p-Xylene Isomer [106-42-3]	100 ppm 150 ppm	8- and 12-hour TWA 15-minute TWA	1989	(NC 1989)* (D 1989)* (R 1989)*
†2,6-Xylidine [87-62-7]	0.5 ppm	8-hour TWA	1988	C-A 1983
Zaclon® Galvanizing Fluxes		Not currently in use within DuPont. See the List of Inactive AELs for details.		
Zepel® 6700	1 mg/m³	8-hour TWA, respirable dust	1990	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Zepel® 7040	1 mg/m³	8-hour TWA, respirable dust	1990	
Zepel® Fluoromonomer		See Zonyl® TM		
Zinc Chloride [7646-85-7]	1 mg/m³	8- and 12-hour TWA	1990	
†Zinc Chromate [13530-65-9]	0.05 mg/m³	8-hour TWA, as chromium	1992	C-H 1975
Zinc Cyanide [557-21-1]	7 mg/m³	8-hour TWA, skin	1990	
Zinc Phenyl-phosphinate [25070-22-8]	1 mg/m³	8-hour TWA	1990	
Zinc Phenyl-phosphonate [34335-10-9]	1 mg/m³	8-hour TWA	1990	
Zineb [12122-67-7]	2 mg/m³	8-hour TWA	1991	
Zonyl® BA [65530-60-1]	5 mg/m³	8-hour TWA	1990	
	15 mg/m³	15-minute TWA		
†Zonyl® TBS [80010-37-3]	1 mg/m³	8-hour TWA	1992	
†Zonyl® TELA [25398-32-7]	10 mg/m³	8-hour TWA	1992	
†Zonyl® TM [65530-66-7]	2 mg/m³	8-hour TWA	1992	
Zytel® Nylon (polyamide) [32131-17-2]	10 mg/m³	8-hour TWA, total dust	1988	
	5 mg/m³	8-hour TWA, respirable dust		

FOR DUPONT USE ONLY

INACTIVE AELS

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
t-Butyl Isocyanide [7188-38-7]	0.1 ppm	8-hour TWA, skin	1985	D 1985 R 1985
†Cinmethylin (Used in Cinch® Herbicide) [89368-00-3]	1 mg/m³	8- and 12-hour TWA	1987	(NC 1987) (D 1987) (R 1987)
Cupric Hydroxide [1344-69-0]	1.5 mg/m³	8-hour TWA	1985	
6,7-Dihydro-2-methyl-5H-cyclo-penta(d)pyrimidine [36274-29-0]	0.03 ppm 0.02 ppm 0.1 ppm	8-hour TWA 12-hour TWA 15-minute TWA	1985	
Dimethoxane [828-00-2]	50 ppm	8-hour TWA	1986	
N,O-Dimethyl-hydroxylamine [1117-97-1]	20 ppm	8-hour TWA	1985	
1,12-Dodecane-diamine [2783-17-7]	0.1 mg/m³	8- and 12-hour TWA	1989	
Fenvalerate (Used in Pydrin® Insecticide) [51630-58-1]	2 mg/m³	8- and 12-hour TWA, skin	1988	
Kelthane [115-32-2]	1 mg/m³	8-hour TWA	1985	

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CHEMICAL [CAS#]	AEL	REMARKS	DATE/ STATUS	ELC GUIDELINES
Lindane [58-89-9]	0.5 mg/m³	8- and 12-hour TWA, skin	1990	
o-Nitroaniline [88-74-4]	3 mg/m³ (0.5 ppm)	8- and 12-hour TWA, skin	P1991	
†p-Nitroaniline [100-01-6]	3 mg/m³	8- and 12-hour TWA, skin	P1992	(c 1992) (R 1992) (D 1992)
p-Nitrobenzyl Chloride [100-14-1]	2 ppb 10 ppb	8- and 12-hour TWA Instantaneous Maximum	1984	
m-Nitrochlorobenzne [121-73-3]	3 ppm 1 mg/m³	8- and 12-hour TWA, skin (vapor) and 8- and 12-hour TWA, skin (particulate)	P1992	
Orlon® (Polyacrylonitrile) [9010-78-0] and [25014-41-9]	10 mg/m³ 5 mg/m³	8-hour TWA, total dust 8-hour TWA, respirable dust	1988	
Siduron [1982-49-6]	10 mg/m³	8- and 12-hour TWA, total dust	1992	
2,4-Toluenediamine [95-80-7]	0.25 mg/m³	8-hour TWA ELC Reclassification Pending	1985	C 1975
Zaclon® Galvanizing Fluxes	1 mg/m³	8-hour TWA, as zinc chloride	1985	

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EMERGENCY EXPOSURE LIMITS

CHEMICAL	EEL	TIME PERIOD	CEILING
Ammonia	500 ppm 300 ppm	10 minutes 10-60 minutes	500 ppm
Carbon Monoxide	900 ppm 170 ppm	10 minutes 10-60 minutes	900 ppm
Chlorine	10 ppm 7 ppm 5 ppm	1 minute 1-5 minutes 5-60 minutes	10 ppm
Chloroprene	2000 ppm-min	60 minutes	2000 ppm
Chlorosulfonic Acid	20 mg/m^3 10 mg/m^3	15 minutes 15-60 minutes	20 mg/m^3
1,4-Dichloro-butene-2	120 ppm-min	60 minutes	2 ppm
†N,N-Dimethyl-aniline	400 ppm 100 ppm	10 minute 11-60 minutes	400 ppm
Dimethyl Sulfate	30 ppm-minutes	60 minutes	2 ppm
Fluorobenzene	2000 ppm 1000 ppm	1 minute 2-60 minutes	2000 ppm
Fluorosulfonic Acid	10 mg/m^3 5 mg/m^3	15 minutes 15-60 minutes	10 mg/m^3
Formaldehyde	10 ppm	60-minutes	10 ppm
Halon 2402	500 ppm	15 minutes	500 ppm
HCFC-31	2500 ppm 1000 ppm	1-minute 2-60 minutes	2500 ppm
HCFC-123	1000 ppm	2-60 minutes	2500 ppm
Hexafluoro-propylene	6000 ppm-min	60 minutes	1000 ppm

June 17, 1994

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EMERGENCY EXPOSURE LIMITS

CHEMICAL	EEL	TIME PERIOD	CEILING
HFC-338pcc	2000 ppm 1000 ppm	1-minute 2-60 minutes	2000 ppm
HFC-43-10	2500 ppm 1000 ppm	1-minute 2-60 minutes	2500 ppm
High-Boiling Fluorocarbon Liquid	25 mg/m³-min	60 minutes	25 mg/m³
Hydrogen Bromide	35 ppm 20 ppm	1-10 minutes 11-60 minutes	35 ppm
Hydrogen Chloride	35 ppm 20 ppm	1-10 minutes 11-60 minutes	35 ppm
Hydrogen Cyanide	500 ppm-min	50 minutes	100 ppm
Hydrogen Fluoride	200 ppm-min	60 minutes	100 ppm
Hydrogen Sulfide	100 ppm 50 ppm 25 ppm	1 minute 2-10 minutes 11-60 minutes	100 ppm
Maleic Anhydride	1 ppm	60-minutes	1 ppm
Methylamine	500 ppm 300 ppm	10 minutes 10-60 minutes	500 ppm
2-Methyl-1,4-butanediol	2000 ppm-min	60 minutes	1000 ppm
Methylene Chloride	800 ppm	60 minutes	800 ppm
Methyl Isocyanate	4 ppm-min	60 minutes	4 ppm
3-Methyl-tetrahydrofuran	1000 ppm	60 minutes	1000 ppm

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EMERGENCY EXPOSURE LIMITS

CHEMICAL	EEL	TIME PERIOD	CEILING
Pentafluoro-propionyl Peroxide	25 ppm-min	60 minutes	1.5 ppm
Perfluoroiso-butylene	6 ppm-min	60 minutes	2 ppm
Phosgene	10 ppm-min	60 minutes	2 ppm
Sulfur Dioxide	5 ppm 3 ppm	1-10 minutes 11-60 minutes	5 ppm
Sulfuric Acid	20 mg/m³ 10 mg/m³	15 minutes 16-60 minutes	20 mg/m³
Tetrafluoro-ethylene	100,000 ppm-minute	60 minutes	20,000 ppm
Tetrahydrofuran	1000 ppm	60 minutes	1000 ppm
Titanium tetrachloride	500 mg/m³-minute	60 minutes	500 mg/m³
Trimethylamine	500 ppm 300 ppm	10 minutes 10-60 minutes	500 ppm

FIRE EMERGENCY EXPOSURE LIMITS (FEELS)

†HFC-23 (Trifluoromethane)	230,000 ppm 200,000 ppm	1-minute 2-15 minutes
†HCFC-124 (2-Chloro-1,1,1,2-tetrafluoroethane)	10,000 ppm	15-minute ceiling

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COMMUNITY EXPOSURE GUIDELINES

Airborne Guidelines

DS
END

CHEMICAL	CEGa
Acetic Acid	- 1 ppm
Acrylonitrile	- 20 ppb
Ammonium Perfluorooctanoate (C-8)	- 0.0003 mg/m³
Benzene	- 0.05 ppm
1,3-Butadiene	- 0.1 ppm
Carbon Tetrachloride	- 0.1 ppm
Carbonyl Sulfide	- 0.2 ppm
Chlorine	- 0.05 ppm
Chloroform	- 0.2 ppm
Chloroprene	- 0.5 ppm
Dibromomethane	- 1 ppm
Dimethylacetamide	- 0.4 ppm
Dimethylformamide	- 0.4 ppm
1,4-Dioxane	- 1 ppm
Dodecanedioic Acid	- 0.5 mg/m³ (total dust)
Ethanolamine	- 0.3 ppm
Ethylene Dibromide	- 2 ppb
Ethylene Dichloride	- 0.1 ppm
FC-116 (Hexafluoroethane)	- 100 ppm
Formaldehyde	- 0.2 ppm
1,4-Hexadiene	- 0.5 ppm
n-Hexane	- 5 ppm
HFC-23 (Trifluoromethane)	- 100 ppm
HFC-125 (Pentafluoroethane)	- 100 ppm
Hydrogen Chloride	- 0.5 ppm
Hydrogen Cyanide	- 2 ppm
Hydrogen Fluoride	- 0.1 ppm
Maleic Anhydride	- 0.01 ppm
Methyl Chloride	- 2 ppm
Methylene Chloride	- 10 ppm
3-Methyltetrahydrofuran	- 10 ppm
Nitrogen Dioxide	- 0.2 ppm (60-minute TWA) in combination with the National Ambient Air Quality Standard of 100 ug/m³ (0.053 ppm) - Annual arithmetic mean

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June 17, 1994

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COMMUNITY EXPOSURE GUIDELINES

Airborne Guidelines

Nitrous Oxide	- 5 ppm
Norbornadiene	- 5 ppm
Pentafluoroallyl Chloride	- 5 ppb
Phosgene	- 0.01 ppm
Sulfur Dioxide	- 0.2 ppm (60-minute TWA) in combination with the National Ambient Air Quality Standards of 80 ug/m^3 (0.03 ppm) - Annual arithmetic mean and 365 ug/m^3 (0.14 ppm) - 24-hour concentration not to be exceeded more than once per year
Tetrachloroethylene	- 1 ppm
Tetrafluoroethylene	- 2 ppm
Tetrahydrofuran	- 10 ppm
Titanium Tetrachloride	- 0.02 mg/m^3

Drinking Water Guidelines

CHEMICAL	CEGw
Ammonium Perfluorooctanoate (C-8)	- 1 ug/L
Carbon Disulfide	- 0.8 mg/L
CFC-11 (Trichlorofluoromethane)	- 10 mg/L
CFC-113 (1,1,2-Trichloro-1,2,2-trifluoroethane)	- 10 mg/L
Chloroform	- 0.1 mg/L
Dimethylacetamide	- 2.5 mg/L
Dimethylformamide	- 2.5 mg/L
1,4-Dioxane	- 9.0 mg/L
†HCFC-123 (2,2-Dichloro-1,1,1-trifluoroethane)	- 3 mg/L
HMPA (Hexamethylphosphoramide)	- 0.0002 mg/L
Triethylene Glycol	- 10 mg/L

AEL30.5

WJB000071

June 17, 1994

DUPONT

September 28, 1994

DuPont Confidential

TO:
J. P. GLAS - B13232, WILM.
P. V. TEBO - N2514, WILM.
R. L. BAILLIE - B168
B. L. HUDSON - B168
P. K. MATHUR - B168
H. D. RAMSEY - B1
H. BENJAMINS - DORDRECHT
C. S. SERINGER - B13256
B. DAENGELI - GENEVA
C. W. DIETZ - CRP 711
D. A. HOLMES - CRP 711
J. M. KEEGAN - CRP 711
K. KIMPEL - GENEVA
J. B. PORTER, JR. - B13246
R. P. ROGERS - MDF, JAPAN
M. E. STOOKEY - B13236
V. RICE - D8082, LEGAL
W. E. BACHMAN - CRP 711
J. P. BOLLMEIER - ESL 272
R. A. BRANDENBURG -DW
P. W. BRITT - D11100-2
G. L. KENNEDY - HASKELL
N. MARINI - CH. WKS
W. H. MARTIN - CRP 713
F. P. MULHERIN - B13362
A. J. PLAYTIS - B24
R. L. RITCHEY - CRP 711
E. K. SAKATA - CH. WKS
T. J. SCHROOTS - GENEVA
W. M. STEWART - B1
D. N. WASHBURN - CRP 711
K. YOKOYAMA - MDF, JAPAN
R. J. ZIPFEL - B21
B. E. SMART - E328
A. E. FEIRING - E328
S. V. GANGAL - E269
A. C. SOBRERO - DORDRECHT
R. D. STARK - GENEVA
J. C. MOORE - RICHMOND
D. L. PEET - CRP 711

FR: SHARON BOONE - WASHINGTON LABORATORY

C-8 AMMONIUM PERFLUOROOCTANOATE FLUOROSURFACTANT

STRATEGIES AND PLANS

Attached is your numbered copy of the "C-8 Ammonium Perfluorooctanoate Fluorosurfactant, Strategies and Plans" document. The initial draft of this document was titled "White Paper, C-8 Ammonium Perfluorooctanoate Fluorosurfactant." Please return your draft copy to me for destruction.

Please return this document when you no longer need it.

Attachment

EXHIBIT
14
Kennedy

½ life - stab 1½-4 yrs

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RJZ029978

C-8
AMMONIUM PERFLUOROOCTANOATE FLUOROSURFACTANT

STRATEGIES AND PLANS

R. J. Zipfel, et al

September 15, 1994

Copy No. ____

C-8 Ammonium Perfluorooctanoate Fluorosurfactant

TABLE OF CONTENTS

OBJECTIVES OF REPORT

This summary of programs involving C-8, ammonium perfluorooctanoate fluorosurfactant, is intended to provide the basis for program alignment and to be a touchstone in discussions on the procurement, use, recovery, recycle, and replacement of C-8. Toxicology, personnel exposure and environmental emissions are discussed.

BACKGROUND ON USE OF C-8

C-8, ammonium perfluorooctonoate, is the polymerization surfactant used for the manufacture of TEFLON® fine powder, dispersion, FEP, PFA, and micropowder fluoropolymers and VITON® and KALREZ® fluoroelastomers. Its use in DuPont began in 1951. C-8 is purchased from various global suppliers including 3M, Miteni, and Hoechst. Historically, it had been received as a dry powder. However, to reduce employee exposure to C-8 purchase and use has been shifting to aqueous solutions.

C-8 is mostly unaffected by the manufacturing processes. This results is most of the C-8 ending up a part of our product (dispersions) or as a waste to the environment.

Global usage is:

	Amount (lb)		
Site	1991	1992	1993
Washington Works	71,100	67,200	72,000
Dordrecht Works	31,400	37,950	33,280
Shimizu Works	15,950	14,700	20,830
Chambers Works	2,000	1,500	1,954
Experimental Station	200	200	200
TOTAL	120,650	121,100	128,264

PROGRAM GOALS AND STRATEGIES

To continue to manage the use of C-8 and C-8 containing products in a way that exposure to employees and customers continues to be within accepted control levels, and to drive environmental emissions towards zero. To accomplish these goals we have established the following program strategies.

I. Comply with all laws and regulations governing C-8 use and disposal.

II. Keep C-8 exposures below the AEL for employees and the CEG for the general public.

III. Communicate C-8 information to employees and the public in consultation with Site, SBU, External Affairs and Legal.

IV. Reduce environmental C-8 emissions 50% by 1997 vs 1993 base. (See appendix B).

V. Evaluate replacement of C-8 with other less toxic materials.

VI. Choose C-8 suppliers based on meeting our business objectives which include C-8 recovery and recycle.

TOXICOLOGY

A work place dust exposure level of 0.01 mg/m^3 has been established both by the ACGIH TLV Committee and DuPont's AEL Committee (levels to which workers could be exposed 8 hr/day, 5 days/week for a working lifetime without damage to health). The slow clearance of C-8 from human blood justifies the setting of a low permissible exposure. The DuPont CEG of 0.0003 mg/m^3 is based on reductions due to continuous exposure (24 hr/day rather than 8 hr/day; lifetime vs working lifetime) and the possibility of sensitive subpopulations (aged, very young, pre-existing disease, etc.).

C-8 has moderate acute oral and inhalation toxicity in rats, is non-irritating and very low in acute dermal toxicity in rats and rabbits and is moderately irritating to rabbit eyes. C-8 is not a developmental toxin in rats, and is not a genetic toxin by the Ames test. C-8 was found to not bio-accumulate in fish.

The half-life in the human blood system appears to be 1½ to 3 years. No adverse health effects were found in 3M workers or in a study of liver function in DuPont Washington Works

employees. A possible increase in prostate cancers has been reported at a 3M facility making C-8 (among other chemicals). As follow-up to this preliminary finding 3M may look at hormone levels in the exposed population and at genetic toxicity.

Peroxisome proliferation in the rodent liver is being studied so extrapolation to humans can be based on mechanism rather than on safety factor. Rodents are more sensitive than humans to peroxisome proliferators.

See appendix E for details.

PERSONNEL MONITORING

DuPont began extensive employee monitoring for C-8 in 1978 after the 3M Company indicated that organic fluorine was detected in the blood of workers exposed to certain fluorinated surfactants. Personnel air monitoring (defined by Engineering Standard S12T) and blood monitoring were undertaken at the Experimental Station, Dordrecht, Washington and Chambers Works. All these sites are currently in compliance with the DuPont AEL (Acceptable Exposure Limit) of 0.01 mg/m^3 for airborne C-8. Shimizu began area monitoring in 1989 and personal air monitoring in 1993. Analysis of data to statistically verify AEL compliance is not yet complete.

Blood monitoring has defined initial blood concentrations, the relationship to airborne and skin exposure, and the blood concentration decay rates when exposure is eliminated or reduced. Annual blood monitoring at Washington Works was changed in 1990 to a recommended five year frequency because of the long half-life of C-8 in the human blood system; the next major round is due in 1995. See Appendix D for additional information. Blood sampling is available annually if desired by an employee.

INDUSTRIAL HYGIENE AND PERSONNEL PROTECTION

Engineering and administrative controls to reduce exposure include: Fine Powder dryer C-8 abatement scrubbers at Shimizu and Washington Works, FEP torus disc dryer vent scrubbers at Dordrecht and Washington Works, use of liquid C-8 water solutions at all sites. (Dordrecht converting 1Q'95), sealing of leaks to the work place, and restricted work zones (where the AEL is exceeded) requiring breathing protection. Personal protective equipment against airborne exposure and skin contact has been specified.

EMPLOYEE AND CUSTOMER NOTIFICATIONS

Material Safety Data Sheets (MSDS) notify employees and customers of the health effects

of using or processing materials. Extensive processing and handling information is provided in literature, including the Safety In Handling Guide published by the Society of Plastics Industry. New employees are given an initial detailed orientation on the hazards of the materials they work with and annual reviews thereafter. Changes in hazards are reviewed promptly. MSDS are sent to new customers and to all customers when updated. If a change in potential health effects is large, or involves a carcinogen, communication is face-to-face followed with a cover letter and the new MSDS.

C-8 IN THE ENVIRONMENT

In 1993, C-8 emissions at Washington Works, Dordrecht, Shimizu, Chambers Works, and Experimental Station were:

Destination	lb/yr
Water	73,319
Air	21,930
Landfill	5,630
Product to Customers	14,190
Decomposed	5,795
Unaccounted	200
Recovered	7,200
TOTAL	128,264

C-8 is released into the Ohio, Delaware, James and Merwede Rivers, and Sugura Bay. C-8 is found in the groundwater below the Dordrecht and Washington Works sites and at low levels (below the 1 ppb CEG_w) in the Parkersburg area Lubeck Public water system and in the water supplying the sanitary water to the Washington Works site. C-8 levels in these waters are all below the CEG of 1 ppb (Community Exposure Guide; see Appendix C for the definition) except that Washington Works groundwater has 2-3 ppb.

C-8 has been found in the surface and ground waters around the landfills used by Dordrecht and Washington Works. The Letart landfill, primary landfill at the Washington Works, is scheduled to close at the end of 1995. C-8 containing materials are no longer placed in the other two landfills used by Washington Works.

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C-8 is decomposed to a hydride by high temperature (~300°C) pyrolysis.

SOIL REMEDIATION: ELECTROOSMOSIS STUDIES

The soil beneath the Parkersburg "Supernate" pond location is contaminated with C-8. This has been reported to the EPA as part of the site's RCRA Corrective Action Permit for its solid waste management units (SWMU). In anticipation of having the EPA order the site to remediate the supernate pond SWMU, the site chose to investigate what practical alternatives were available to the more costly method of excavation and incineration. One of these methods was the use of electroosmosis technology being developed at the University of Delaware.

Laboratory studies indicate C-8 can be removed from soils by more than 90% by electroosmosis (EO) provided pH is controlled. The practicality of in-situ EO to remediate the upper 10-12 feet of soil is unknown. As field experience is gained (DuPont is conducting a field pilot at Spruance for HMPA), the suitability of EO versus containment can be better assessed.

RECOVERY TECHNOLOGY AND PATENTS

Scrubber on Washington Works (WW) Fine Powder dryers to decrease air emissions has not demonstrated design recovery rates. Basic data is being developed to increase recovery from air emissions at WW and design units for Dordrecht (DW) and Shimizu (SW). Completion of technology development to recover C-8 from air and supernate expected October, 1994. Hoechst asserts they have scrubbing technology and aqueous recovery technology. Hoechst patent US 4,369,266 (issued January, 1983) discloses, but does not claim, surfactant recovery from filtrate (or supernate); only surfactant recovery from dispersions is claimed. 3M has purified recovered C-8 from WW and steam distillation has been demonstrated at ESL. FEP may be able to use recovered C-8 without purification.

REPLACEMENTS

Search for C-8 replacements date back to 1979. The initial efforts indicated that ZONYL® TBS was the best potential candidate (ref: PPD 86-W-3, Improved Dispersing Agents for Fluoropolymerizations). Initial use of ZONYL® TBS was in 1986 in the FEP process. Use of the ZONYL® TBS grew to 25% of the FEP product line, but has since been reduced to less than 10% due to operational difficulties.

Current replacement efforts are focussed on the use of ZONYL® 6,2 TBS, and scouting efforts by CS&E at the Experimental Station. ZONYL® 6,2 TBS has shown some promise in

semiworks testing for FEP and ptfe dispersion products. CS&E is currently examining new surfactant materials specifically for the FEP product line and looking for a universal (all product types) replacement for C-8.

PROGRAMS

A listing of current and proposed 1994 programs for C-8 related matters is in Appendix A. Some of these programs provide alternate paths to achieve the same objective. Appendix B presents a five year road map of key environmental milestones

PURCHASING STRATEGY AND PLAN

DuPont has a global price-volume arrangement with 3M. Miteni is a second source for Dordrecht to insure competitive pricing; purchases should be based on opportunistic pricing while maintaining their global DuPont share at 10%-20%. The use of recovered C-8 (RC-8), costing 66% less than virgin material, at Washington Works should be increased. The price level for discontinuing reclamation work is approximately $3 per pound (wet basis). C-8 recovery programs should use supplier expertise and resources where possible.

WORK WITH SUPPLIERS AND OTHERS

Principles: Use outside resources to leverage efforts, minimize total costs, and achieve business goals as effectively as possible. Be sensitive to possible loss of competitive advantage in the development or use of proprietary technology by working with outsiders.

Hoechst: Would like to be second DW supplier. Is interested in further toxicology work and in partnering on C-8 replacement.

Miteni: Developing liquid C-8. Proposed partner for DW recovery work.

3M: Global price-volume agreement. Have purified recovered C-8 for WW. Proposed partner for C-8 recovery from supernate and FEP coagulator effluent at WW. Partnering on analytical.

APPENDIX A

1994 C-8 PLAN

A. Personnel Monitoring

1. Review first year of C-8 personnel monitoring at Shimizu.

   Timing: October 1994

2. Decide on blood sampling program for Dordrecht and Parkersburg.

   Timing: October 1994

3. Audit all sites for compliance to MSDS and DuPont AEL (including contractors).

   Timing: December 1994

4. Complete C-8 communications with C-8 affected personnel at Dordrecht.

   Timing: Complete

B. Toxicity Tests

1. Complete study on C-8 accumulation in fish.

   Timing: Complete

2. Complete peroxisone proliferation study of C-8 and other chemicals.

   Timing: December 1994

C. Groundwater Contamination

1. Complete cost analysis for groundwater remediation at Parkersburg.

   Timing: December 1994

2. Determine if current chlorocarbon groundwater treatment facility at Dordrecht will be adequate for C-8.

Timing: December, 1994

3. Determine extent and assess risk of groundwater contamination around Dordrecht C-8 containing landfills.

Timing: Determine sampling plans October - 1994
Complete analysis - January 1995

D. Personnel Exposure

1. Implement use of liquid C-8 at Dordrecht.

Timing: March 1995

2. Alter PTFE dispersion post C-8 addition procedure at Parkersburg.

Timing: Complete

3. Improve efficiency of existing scrubbers in Dordrecht FEP/VITON® on C-8 containing gas streams.

Timing: July 1995

4. Consider auditing customers for the proper handling of PTFE and FEP dispersions, and disposition for C-8 containing wastes.

Timing: October 1994

E. C-8 Toxicity Communications

1. In-depth toxicology communications with 3M, Hoechst and DuPont.

Timing: Complete

2. Have in-depth C-8 toxicity discussions with affected personnel at Shimizu.

Timing: 2H, 1994

F. C-8 Alternatives

1. Demonstrate ZONYL® 6.2 TBS in FEP

   Timing: First plant batch - Complete
   Multiple batch test - TBD

2. Develop in conjunction with Finishes ZONYL® 6.2 TBS recipe for PTFE dispersions.

   Timing: Semiworks testing - Complete
   Plant test - December 1994

3. Develop other alternatives to C-8.

   Timing: Reactor facilities for FEP at ESL - Complete
   Demonstrate FEP poly - Complete
   Demonstrate mixed surfactant concept - October 1994
   Prepare polymer samples for evaluation - November 1994
   Toxicity testing of alternatives by Haskell - March 1995

G. Improve Efficiency of Fine Powder C-8 Recovery Facilities at Parkersburg

1. Install spray quench into scrubber feed line.

   Timing: Complete

2. Add additional heat to scrubber to reduce mist formation.

   Timing: 1st Phase - Complete
   2nd Phase - October 1994

3. Upgrade operation of ion exchange beds.

   Timing: Complete

4. Complete material balance of scrubber.

   Timing: Resolve sampling problems - Complete
   Complete balance - Complete
   Reaffirm balance - September 1994

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5. Test Parkersburg recovered C-8 material directly in FEP plant.

Timing: TBD

6. Purify recovered C-8 by steam distillation at ESL.

Timing: Distillation - Complete
Concentration - September 1994

H. Utilize C-8 Suppliers To Assist in the Development of C-8 Recovery Technologies

1. Send concentrated C-8 scrubber material to Miteni to develop Dordrecht fine powder recovery process.

Timing: Samples Sent - Complete
Final Results - December 1994

2. Send supernate samples to 3M for development of C-8 recovery technologies.

Timing: Dilute sample - Complete
Concentrated sample - TBD

3. Establish secrecy agreement with Hoechst for C-8 recovery from supernate.

Timing: Complete

4. Send concentrated FEP effluent sample to 3M for development of C-8 purification from in-situ surfactant or, conduct separation tests in-house (WW or ESL).

Timing: Sample concentration - Complete
In-house testing - October 1994

5. Qualify Hoechst C-8 at Dordrecht.

Timing: T-637 is complete.
Rest of production line - TBD.

I. Develop Scope to Reuse C-8 From Supernate

1. Demonstrate feasibility to separate C-8 from "Triton" using reverse osmosis technology.

   Timing: September 1994

2. Develop basic data for ion exchange separation of C-8 and "Triton".

   Timing: October 1994

3. Technology exchange with Hoechst.

   Timing: TBD

4. Reduce quantity of supernate shipped to Chambers Works for disposal.

   Timing: IVQ95

5. Work with Tetra to install recovery facilities at Parkersburg.

   Timing: Decided to not pursue recovery using Tetra facilities.

J. Eliminate Solid Waste Shipments to Letart Landfill

1. Institute metal reclaim program for TEFLON® containing metal.

   Timing: Locate acceptable metal reclaimer - Complete
   Complete program - December 1994

2. Reclaim C-8 containing FEP fluff for sale.

   Timing: Establish contract with Ohio Valley B&B for reclaim - December 1994.
   Recover all FEP waste fluff - April 1995

3. Reclaim PTFE from coagulum for sale.

   Timing: Complete basic data on C-8 removal - Complete
   Establish contract with reclaimer - October 1994
   Recover all PTFE from coagulum - January 1995

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4. Establish procedures to landfill TEFLON® waste at the Dry Run Landfill in West Virginia.

Timing: December 1994

5. Eliminate use of fine powder dryer paper.

Timing: Parkersburg No. 2 drier - 1Q95
Parkersburg No. 3 drier - 3Q95
Dordrecht drier - 4Q95

K. Recover C-8 From Customer Waste Streams

1. Initiate C-8 recycle and recovery from U. S. Gore.

Timing: Initial shipments - TBD

L. Complete technology development for Dordrecht Fine Powder C-8 recovery system.

Timing: December 1994

M. Develop global C-8 Management Plan

Timing: 1Q95

RJZIPFEL:ssb
4/25/94
Rev. 9/2/94
Rev. 9/27/94

APPENDIX B

C-8 MANAGEMENT

5 YEAR ROAD MAP

FPE Implementation Roadmap May-94

SUB-STRATEGY	TIME / WHO	94	95	96	97	98	ISSUES/NOTES
G. Responsible Care	JBP RLR CSS	o Develop 5-road maps for priority issues	o Monitor & remap	→→→→	→→→→	→→→→	
		o Drive priority issues	→→→→	→→→→	→→→→	→→→→	
4. C-8 Management	RLR RJZ	o Quantify global SBU goal for emission reduction					
	RJZ	o Develop material balance globally					
	RJZ	o Demonstrate drier recovery system objectives -WW					
	RJZ	o Basic data-C-8 recovery fr. supernate					Key is Triton separation
Stakeholders & Expected Results		Employees - reduced exposure Community - reduced exposure Stockholders- improved earnings through lower costs, higher productivity Customer - improved product properties					

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F.. Implementation Roadmap May-94

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RJZ	o Explore supplier recovery of C-8 fr. FEP effluent					
	RJZ	o Test 6,2 TBS in FEP plant - WW					
	MG	o Develop & plant test Zonyl 6, 2TBS recipe for PTFE dispersion					
	BES PHL RV	o Screening tests on alt. surf. toxicity					
	BES	o Evaluate alt. surfactants and samples for polymer characteriza-tion					
Stakeholders & Expected Results							

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RJZ0299S

FPE Implementation Roadmap May-94

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RAB	o Define Miteni recovery capability					
	KK	o Establish secrecy agreement with Hoechst					
	RAB	o Implement solution C-8 DW FEP/PTFE					
	RAB	o Withdraw & purify groundwater at DW site					
	RAB	o Eliminate known sources of potential groundwater contamination - DW	→→→→				
	RAB	o Define scope and path forward for any ground-water investigation at landfills used by DW					
Stakeholders & Expected Results							

EID112816

RJZ029996

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RJZ	o Develop options for reducing C-8 in aqifer below WW					Anticipate EPA to require remediation at former supernate ponds.
	RJZ	o Pilot recycle of C-8 fr. U.S. customers					
	KML	o Complete study of C-8 accum. in aquatic animals					
	FPM AJP	o O/H exchange visits w/3M-U.S.					
	GLK	o Initiate 3-party toxicology exchange w/ Hoechst/3M					
	FPM	o TLV lowering implementation/commun.					
Stakeholders & Expected Results							

EID112817

RJZ029997

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RV FPM	o Perox. prolif. testing wrap-up and follow-up					
	FPM RJZ	o Audit C-8 users for MSDS, ppe, monitoring - Chambers - Shimizu	→→→→ - Dordrecht	→→→→ - Parkersburg	→→→→ - Chambers - Shimizu	→→→→ - Dordrecht	
	RJZ RLR	o Develop/present commun. package for Shimizu					
	RLR RDS RJZ	o Incr. awareness of tox. issues w/Prod. Stewards					
	RLR RJZ		o Revisit goals and material balances	→→→→	→→→→	→→→→	
	RJZ BES RAB RLR		o Decision on C-8 alternative				
Stakeholders & Expected Results							

EID112818

RJZ0299 98

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RLR SL		o PMN/TSCA filings for alt. surf.				
	RJZ		o Plant test C-8 alt. at WW FEP, PTFE				
	RJZ		o Continuous Fine Powder Dryer Belts - WW				
	RJZ		o Implement C-8 Alt. at WW FEP, PTFE				
	RJZ		o VITON effluent re-covery basic data				
	RJZ		o Reduce land-filling of C-8 by 90%-U.S.				
	RJZ GW		o Close Letart landfill				
Stakeholders & Expected Results							

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
	WHO						
4. C-8 Management (Continued)	RJZ RAB RDS		o Eval. customer base for take-back opport.				
	RAB		o Improve scrubber efficiency, DW FEP				
	RAB		o Continuous fine powder belts - DW				
	RJZ			o Reduce C-8 from WW FEP by 95%			
	RJZ			o Install dryer recovery facility; Reclaim C-8 - SW			
	RAB RJZ			o Impl. C-8 alt. at DW FEP, PTFE; SW PTFE			
	RAB				o Install dryer recovery facility; Reclaim C-8 - DW		
Stakeholders & Expected Results							

EID112820

RJZ03000

E Implementation Roadmap May-94

SUB-STRATEGY	TIME	94	95	96	97	98	ISSUES/NOTES
4. C-8 Management (Continued)	WHO RLR RDS RJZ RAB				o Refresher session with Prod. Stewards	o Reduce land-filling of C-8 by 90% - DW	
Stakeholders & Expected Results							

EID112821

RJZ03000I

APPENDIX C

APPENDIX C

A Community Exposure Guideline ("CEG") is an exposure guideline established by Haskell Laboratory. The CEG assumes a 24-hour lifetime exposure by all, including the most sensitive individuals, in an exposed community population. Exposure above the CEG will not necessarily result in any adverse effects. Where data indicates that the CEG may be approached or exceeded, Haskell, the appropriate Business and Legal will evaluate, what action, if any should be taken. It is the Company's intent to maintain exposure below the CEG.

APPENDIX D

APPENDIX D

March 19, 1990

TO: J. G. LOSCHIAVO - FIBR - SPRUANCE
R. D. LANYON - PPD - WASHINGTON WORKS
W. E. CRAWLEY - PPD - WASHINGTON WORKS

FROM: H. A. SMITH Howard Smith

REVIEW OF WASHINGTON WORKS C-8 DATA
PERSONNEL AIR MONITORING AND BLOOD DATA

Following is a summary of our meeting and the conclusions reached. Please get back to me with comments as soon as possible. I've attached the package of backup data used at the meeting.

C-8 personnel air monitoring data taken over the period April 1988 through September 1989 (220+ samples covering 22 jobs) and all personnel C-8 in blood data going back to 1979-80, were summarized and reviewed. When correlating blood data with air monitoring data by PERS jobs, the only people included in the blood data base were those who had been in the indicated job for years; had not moved all over the Fluoropolymers area; and are still in the jobs. The task of interpreting the data is complicated by the fact that the air monitoring data is recent whereas the blood data (because of the very slow drop off rate of C-8 in the blood) essentially reflects exposure dating back to the "early days".

Following are the conclusions from this review:

1) There is a correlation between C-8 personnel air levels and C-8 in blood levels, and between skin contact and C-8 in blood levels. Table 1 summarizes the blood data/air data/skin contact potential by PERS jobs. It is interesting to note that the jobs that have high C-8 in blood populations and that are not in compliance with the AEL, are those jobs that have potential for C-8 skin exposure. Figure 1 is a plot of personnel air levels for a PERS job code vs. blood levels for the same jobe code. Figure 1 also shows the high blood levels that do not fit the curve which we attributed to skin contact on the part of the specific individuals.

2) Figure 1 would indicate that exposure at the AEL of 0.56 ppb would equate to a blood level of ~3 ppm.

EID112825

RJZ030005

3) Background blood levels, for people just working in this area of the plant, are in the 0.1-0.5 ppm range.

4) The effect of skin contact is strong and apparent as demonstrated in Table 1 and Figure 1, especially documented by jobs 08PE, 08PH, 08PF. Skin contact results in blood levels in the 7-8 ppm range or way higher. This position is also supported by 3M and by review of all the other blood plots we have; where some people stand out with very high blood levels.

5) A side observation - people working in a job seem to jump to the blood level for the job and stay there.

6) Drop-off rate for C-8 in the blood is a half life of ~4-5 years or more. This is based on a very small amount of data on pensioners and on the observation that there is a slight perceived decline in workers in the various jobs.

7) Because of the slow drop-off rate, annual blood testing is telling us nothing. We should discontinue blood testing (unless an employee requests it) for now and reconsider taking another "snapshot" in a few years after we have completed our process improvements (e.g. use of C-8 in solution).

   When we elect to do more blood monitoring, we should sample as many workers as possible. We would need the people new to the area as well as the older workers because the older workers would still be on the slow decay C-8 in blood curve. Even at best the data will again be difficult to interpret because of the frequent job-to-job movement of people within the fluoropolymers area and from area to area on the Washington Works site. Other confounding factors that will continue to add to variability are individual work habits and individual biological responses such as excretion rate and retention in body other than in blood (e.g. fat).

8) Eighteen of the 22 PERS jobs were in compliance with the AEL for C-8. Four jobs were out of compliance or marginal. All four jobs involved potential for skin exposure. Use of C-8 in solution should eliminate the problem for three of the four jobs.

HAS/is
Attachment

APPENDIX E

Ammonium perfluorooctanoate has a moderate acute toxicity with an LD50 in rats of 540 mg/kg orally and lethality is produced following 4 hour inhalation of 800 mg/m3 or more by rats. Following dermal applications, the material is nonirritating and very low in acute toxicity with dermal LD50's of 4,300 mg/kg in rabbits and 7,000 mg/kg in rats. Moderate eye irritation, which persisted for at least 7 days, was seen in rabbits following instillation of the material to the eye. Repeated dermal doses of 20 to 2,000 mg/kg to rats produced liver damage in a dose-related fashion and elevated blood organofluoride levels. The increased blood levels were reduced but still detectable, 47 days following the last exposure. Repeated oral doses to rats and mice produce striking liver enlargement with males responding to a greater extent than females. Liver damage was seen in rats and mice fed 300 ppm with relatively little damage seen at 30 ppm. Monkeys did not tolerate oral doses of 30 mg/kg/day while no effects were produced by 10 mg/kg/day or less. No effects were seen in rats inhaling 1 mg/m3, 6 hr/day, 5 days/wk for 2 weeks. Liver changes were seen at exposures of 7.0 and 84 mg/m3. These effects were reversible although the retention in blood was prolonged. Clearance from the blood of female rats was much more rapid due to the presence of an active secretion of mechanism in the kidney.

In a lifetime feeding study in rats, there was a dose-related decrease in weight gain in either 30 or 300 ppm (approximately 1.5 or 15 mg/kg). The primary pathologic changes were in the liver consisting of increased liver weights, increased cell size with vacuolated cytoplasm, and some evidence of hepatocellular degeneration with occasional signs of necrosis. The incidence of benign testicular cancers (Leydig cell adenomas) was increased at 300 ppm but not at 30 ppm. This finding has been repeated and the mechanism appears to be chronic, low level testosterone changes which are induced in a dose-dependent fashion and have a threshold effect level.

Ammonium perfluorooctanoate is not a developmental toxin in the rat at doses up to 25 mg/m3 by inhalation or 100 mg/kg/day orally. The chemical is not a genetic toxin when assayed by the Ames Salmonella typhimurium strains TA1535, TA1537, TA1538, and TA100. It was nonrecombinogenic in the Saccharomyces cerevisiae yeast assay. In the C_3H 10T1/2 colony cell line transformation assay, ammonium perfluorooctanoate showed no evidence of cell transformation.

The concern around the long term effects of ammonium perfluorooctanoate is related to its persistence in human blood. The half-life appears to be 3 to 4 years although the absence of precise data makes this value an estimate. No adverse health effects attributable to exposure were found in 3M workers or in a study of liver function in DuPont Washington Works employees. A workplace exposure levels of 0.01 mg/m^3 has been established both by the ACGIH TLV\ Committee and DuPont's AEL Committee (levels at which workers could be exposed 8 hr/day, 5 days/week for a working lifetime without damage to health). This is based on the absence of liver damage in rodents following inhalation (1 mg/m^3 is the no-observed-adverse-effect level) and by feeding (10 ppm is the no-observed adverse-effect

level). Exposures at the recommended level would be approximately 1000 times less than the exposures producing minimal liver effects. The slow clearance of ammonium perfluorooctanoate from human blood highlights the need for a relatively low permissible exposure. Since the liver effect of C-8 can be produced in rats following relatively low exposures, skin contact should be minimized. A skin notation in both the TLV and AEL should be added to call this to the attention of the industrial hygienist. The DuPont Community Exposure Guideline of 0.0003 mg/m3 (24-hour TWA) is based on the same data but involves additional reductions due to continuous exposure (24 hr/day rather than 8 hr/day; lifetime vs working lifetime) and the possibility of sensitive subpopulations (aged, very young, pre-existing disease states, etc.).

Investigations into the molecular bases for action continue. The first response in rodents, peroxisome proliferation in the liver, is being studied so that the extrapolation to man can be based more on mechanism than on safety factor. As an example, rodents are very sensitive to peroxisome proliferators, man appears less sensitive. This relationship needs to be better quantified. A possible increase in prostate cancers has been reported at a 3M facility making ammonium perfluorooctanoate (among other fluorochemicals). This finding is preliminary and follow-up is being planned. 3M is considering looking at hormone levels in the exposed population. Other ongoing activities include studying environmental effects such as possible bioaccumulation in fish. The genetic toxicity profile is also being expanded by 3M who are considering conducting additional studies.

APPENDIX F

C-8 WHITE PAPER CONTRIBUTORS

WYNNE BACHMAN

RIK BRANDENBURG

GERRY KENNEDY

TONY PLAYTIS

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ROBERT D. SMITH

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R. A. BRANDENBURG	DW	20
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G. L. KENNEDY	HASKELL	22
N. MARINI	CH. WKS	23
W. H. MARTIN	CRP 713	24
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S. V. GANGAL	E269	36
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J. C. MOORE	RICHMOND	39
D. L. PEET	CRP 711	40
LIBRARY	WASHINGTON WORKS	41

DRAFT

Proposal to Conduct a
General Human Health and Environmental Effects
Risk Analysis on C-8

Purpose: The purpose of this project is to evaluate the risks to human health and the environment from exposure to C-8 during manufacture, transport, product use, and disposal of C-8. The analysis will be conducted in a fashion that will provide semi-quantitative estimates of risks so that exposures yielding the highest risks can be identified and recommendations on reducing these risks can be developed. Risks from manufacture, transport and product use will developed in a way that will facilitate future comparisons of risks estimated for potential C-8 alternatives. The project will be conducted in three parts. The first two will be conducted in parallel, in which human health and ecological risks will be characterized. The final part will develop conclusions on exposures that contribute the highest risks so that recommendations for risk management strategies and alternatives can be developed. The project is estimated to take 12 months to complete from the time of initiation. The Exposure analyses listed below will require collaboration with appropriate plant personnel. *(The dates presented assume a Feb. 1, 1997 SBU approval date.)*

Scope:

I. Human Health Risk	Time Line	Est. Cost($)
A. Hazard Identification	4/18/97	8000

Hazards to human health will be reviewed and summarized in this section. The critical toxicity endpoints of relevance to human health risk will be identified and potential dosimeters to be used for interspecies extrapolation of risk will be discussed. The Haskell toxicity summary will be updated as part of this task.

B. Dose-Response Analysis — 9/30/97 — 43,200

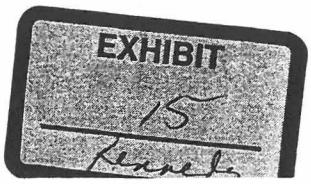
The dose-response characteristics of C-8 will be evaluated. This may include conducting benchmark dose analyses to identify no-observed adverse effect levels where necessary. Appropriate dosimeters for interspecies extrapolation will also be developed based on the likely mode of action. The pharmacokinetics of C-8 will also be reviewed. If possible, rudimentary physiologically-based pharmacokinetics approaches will be developed to facilitate interspecies extrapolation of risk. Risks vs. dose relationships will be developed in this phase

C. Exposure Analysis — 9/30/97 — 16,000

Reasonable exposure scenarios for C-8 will be developed. This are likely to include airborne, drinking water, dermal, and other oral ingestion pathways. Intake rates and durations of exposure will be developed. Haskell will work with an assigned person(s) from the plant site to help characterize these exposure pathways for manufacturing, transport, product use, and waste disposal operations. The business will provide Haskell with data on concentrations of C-8 in the affected media (air, water, soil). These data will be tabulated. Monte Carlo techniques may be used to calculate expected upper confidence limits for these exposures, depending the availability of data. The cost associated with this task include only Haskell personnel time.

D. Risk Characterization — 12/15/97 — 16,000

Risks will be summarized according to the major routes of exposure (air, water, dermal, other oral) for each C-8 application (manufacture, transport, product use, disposal). The risks will be characterized by comparing the likely exposure concentrations to the dose-response relationship. This method is generally referred



to as a Margin of Exposure. The characterization will provide the risk manager with information that will help identify the operations and exposure pathways that present the highest risk. The characterizations will also enable future comparisons to be made of potential risks posed by C-8 alternatives.

II Ecological Effects
- A. Hazard Identification
- B. Dose-Response Analysis
- C. Exposure Analysis
- D. Risk Characterization

III. Recommendations on Risk Management Strategies and Alternatives

12/15/97 4800

This section will evaluate collectively the risks identified to human health and ecological receptors. Based on these analyses, recommendations will be made as to which operations could be targeted to reduce the largest risks for the least cost. This will be a very subjective exercise (narrative) and will require some input from the plant people.

C-8 PRIMATE TESTING

1. We know the mechanism of action for C-8 in the rat - it is a peroxisome proliferator. It is known that man and primates are low to non-responders to peroxisome proliferators.

   Expectation: man/monkey response to C-8 may be different than the rodent.

2. We know the primate response to peroxisome proliferating drugs such as gemfibrozil, clofibrate, and clobuzarit. Liver toxicity (peroxisome proliferation) of these drugs in primates is low. Male and female primates show similar response to these examples as far as overall toxicity is concerned.

   Expectation: significant male/female differences in toxicity would be unexpected.

3. Response to C-8 in the primate, (Long and Griffith) at least in terms of gross tolerance (lethal or no) appears similar.

   Expectation: male = female for lethality

   Overall Expectation: We would not expect sex differences in the primate response to C-8

4. Control of emesis is a necessity in the study proposed.

ICI CHEMICALS & POLYMERS LTD
OCCUPATIONAL HEALTH
THE HEATH
RUNCORN

Notes of a meeting of an ad-hoc group of toxicological representatives of APFO producers and users to discuss forward plans for toxicological research on APFO.

Held at the Hoechst Toxicology Laboratory, Hattersheim - 18 April 1997.

Present:
DG Farrar - ICI C&P Ltd (Chairman)
R Jung - Hoechst Marion Roussel
G Kennedy - DuPont Haskell Lab.
P Lieder - 3M, St Paul

Guests:
CR Elcombe - Biomedical Research Centre, University of Dundee
T Le Duc - 3M, Zwijndrecht
D Mitterberger - Dyneon

Apologies:
M Mistrigio - Miteni

Introduction.

DGF reviewed the history of the ad-hoc group, which had last met in Anaheim USA in February 1996. He explained that, in the past, the group had met under the broad banner of APME although, as not all attending companies were members of APME, it was not a formal sub-group of the APME PTFE Committee.

3M signalled their intention to seek "Associate membership" of APME in order to formalise their involvement in the activity. Miteni should be encouraged to seek similar status.

ACTION - DGF

Update on "APME" proposals.

Dr Elcombe explained the change in his personal circumstances. He reviewed the content of his proposal for further mechanistic studies on APFO which had formed the basis of the Zeneca CTL proposal for work. The project embraced three elements of study:

GK001845

GK001845

1. Effect of APFO on pancreatic acinar cell function, proliferation and differentiation, to include the profiling of bile acids using HPLC and an assessment of its mutagenicity in Ames assays.

2. Initiation/promotion studies in azaserine-treated rats.

3. In-vitro studies in cultured rat and human pancreatic acinar cells and hepatocytes.

CRE would have the facilities to conduct elements 1 and 3 in his new laboratory. Element 2 would need to be conducted elsewhere eg Zeneca CTL.

GK reminded the meeting that DuPont had deemed it essential for CRE to lead any mechanistic investigation on APFO. They saw his relocation to Dundee as no barrier to proceeding along those lines.

Update on US thinking.

GK informed the meeting of bilateral discussions between 3M and DuPont at which it had been agreed that it would be necessary to proceed with a study in Cynamolgus monkeys to answer questions about the relevance of findings on APFO in rats for human health risk assessment. Copies of draft protocols for a preliminary study and a six month oral study in the Cynamolgus monkey, prepared by Covance (a US Toxicology Contract House), were circulated. The actual duration of the study was unclear, but it was thought that at least six months exposure to APFO would be required to make it a valid study.

The species had been chosen because of recent experience by DuPont Haskell Lab of an 18-month year study in rats on the peroxisome proliferator, Wyeth 14,643 (preliminary results of which were presented at the US SoT meeting in March 1997), and of a study in Cynamolgus monkeys on HCFC 123, a chemical which causes liver, pancreatic and testicular tumours in the rat, like APFO. The studies were based on the hypothesis derived from work by Gavin et al (Astra Arcus) that the Cynamolgus monkey, like man, does not possess the Cholecystokinin-A receptor subtype in pancreatic cells, whilst the rat does. It is hypothesised that the pancreatic effects of APFO in the rat are mediated by this receptor and are a consequence of its effects on hepatic cell peroxisomes.

DuPont's view was that this study should precede any mechanistic studies in rats but they recognised that it would be necessary to conduct mechanistic studies at some time in order to link the findings in the rat to those in the monkey.

It was argued that, whilst the monkey study in its own right would add valuable information to the data-base on APFO, the hypothesis upon which the primary aim of the study was based, although potentially valid, was weak. There was little direct evidence on APFO *per se* that linked it to the hypothesis. The links were circumstantial, being based on similarities in biological profile with other chemicals. The most important information (ie that on HCFC123) was not in the public domain. GK agreed to make copies of the monkey study on HCFC123 available to the group.

ACTION - GK

GK001846

It was further argued that the monkey study on its own was unlikely to convince European regulators of the lack of relevance of the findings on APFO in the rat for human health risk assessment. The meeting was reminded that a key reason for proceeding with further studies on APFO was to address both classification and labelling concerns and also food contact applications, both of which were governed by proscriptive regulation in the EU.

Proposals for a way forward.

DuPont (GK) proposed that the group proceed immediately with the study in Cynamolgus monkeys and to defer any decision on the proposals from Dr Elcombe. It was believed that the outcome of the study would resolve the issue of whether the rodent carcinogenicity findings were of concern for man. That information was crucial for DuPont in deciding whether they should continue to use the product

3M (PL) stated their commitment to conduct further research to understand the relevance of the rodent tumour findings for human health. They supported the DuPont proposal to proceed with the study in Cynamolgus monkeys.

Dyneon (RJ) supported the proposal from DuPont.

ICI (DGF) were not happy to proceed with the monkey study in the absence of more direct evidence on the mechanism of toxicity of APFO in the pancreas. It was argued that the monkey study would be expensive and, if it were to be conducted, should be based on as strong a hypothesis as possible. It was doubtful whether a UK Home Office Inspector would give a licence to conduct the study on the basis of the current hypothesis - so there were ethical issues to consider. Furthermore, ICI were not convinced that the outcome of the monkey study, if favourable, would be sufficient to convince EU regulatory authorities that APFO did not present a concern for human health due to its carcinogenicity. ICI preferred to proceed with the mechanistic studies proposed by Dr Elcombe.

As a compromise, GK proposed that the monkey study should proceed and that elements 1 and 3 of Dr Elcombe's proposal be funded in parallel This proposal was supported by the consensus although it was recognised that it might be necessary to conduct the initiation/promotion study in the future if third parties were to be convinced that APFO was operating by a promotional mechanism. DGF agreed to present this compromise proposal to the APME PTFE Committee at its next meeting as the consensus proposal of the ad-hoc group.

ACTION - DGF

The meeting discussed the details of the draft protocols of the monkey studies. It was agreed that a teleconference call would be held on Wednesday 7 May 1997 at 9:00am Eastern Standard Time to receive final comments on the protocols. DuPont agreed to initiate this teleconference.

ACTION - GK

GK001847

The likely costs of the revised study programmes were estimated. The monkey study, if it were confined to a 6 month exposure period, was estimated to cost $350k. The revised project to be conducted by Dr Elcombe was estimated to cost £120k ($180k). Both studies, if initiated before the end of Q2, would run into 1998.

Dr Elcombe was requested to submit a costed proposal for studies as agreed before the end of May 1997 to enable it to be presented to the main APME PTFE Committee at its meeting on 19 June 1997.

ACTION - CRE

DuPont/3M were requested to finalise the protocols and have a fully costed proposal from the contract laboratory in the same timeframe.

ACTION - GK/PL

There was no further business.

DG Farrar.
Toxicology Manager

13.05.97.

GK001848

Robert F Pinchot 06/18/99 08:01 AM

To: Timothy S Bingman/AE/DuPont@DuPont, Andrew S Hartten/AE/DuPont@DuPont, Patricia A Westphal/CL/DuPont@DuPont, Rudolph Valentine/AE/DuPont@DuPont, William J Brock/AE/DuPont@DuPont, Gerald L Kennedy/AE/DuPont@DuPont, Robin C Leonard/AE/DuPont@DuPont, John Gannon/AE/DuPont@DuPont, Maryann J Nicholson/AE/DuPont@DuPont, Michael E McCord/AE/DuPont@DuPont, Oscar T Garza/AE/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, John M Migliore/AE/DuPont@DuPont, Susan S Mileti/DuPont@DuPont, Andrea Malinowski/DPL/DUP@DUP, Bernard.J. Reilly/DPL/DUP@DUP
cc: Richard A Bogda/AE/DuPont@DuPont, Ralph G Stahl_Jr/AE/DuPont@DuPont, Guat-Lian C Kreamer/AE/DuPont@DuPont, Carl F Muska/AE/DuPont@DuPont, Robert L Ritchey/CL/DuPont@DuPont, Jackie K Murphy/AE/DuPont@DuPont
Subject: C-8 Workshop

Thank you for arranging your schedules to attend the C-8 Workshop scheduled for June 30 and July 1. We will start at 8:00 both days in the Learning Center at Barley Mill Plaza, Building 20. The room won't be assigned until that day so please look at the meeting board in the lobby on the second floor for the meeting room. We will be finished by 5:00 on Wednesday and have a dinner planned at Buckley's tavern for all those interested on Wednesday evening (if you haven't told Jackie (999-2704) if you will be attending dinner or not, please let her know as soon as possible).

The desired outcomes for the meeting are:

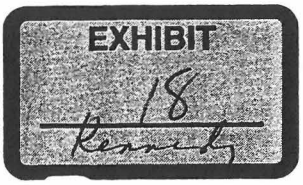
- Understand all that is going on with C-8 studies, risk assessments, etc.
- Develop and Agree to a plan for completing a business risk assessment taking into account the needs of all the stakeholders.
- Understand and Agree to each individual's role in the process.
- Develop data gaps, and plans to fill them. Understand potential synergies among the various groups and develop plan to take advantage of them.

Two days sounds like a lot of time to get these four desired outcomes but with all the people involved, it may take that long. If we are more efficient than Rick and I have planned we may be able to finish early on the second day. The design of the meeting can be made very flexible to fit our needs.

To help you prepare for the meeting, the following is what we need each of you to present at the meeting (for those topics with more than one name, please coordinate among yourselves who will be presenting what):

Legal (Bernie, Andrea): A short summary of the right things to document and not to document.
Business (Mike): An overview of where C-8 goes in our supply chain and other business issues
Specialty Chemicals Issues (Sue): An overview of the fluorosurfactant situation in Spec. Chem
WW RFI (Tim, Andrew, Pat): Detailed time line and data needs to proactively plan for the EPA's reaction to the RFI report
WW Dry Run Landfill (Rudy): Expected timeline and data needs to respond the issues at that landfill
Toxicology (Bill, Gerry, Robin): Expected time lines for the human health risk assessment for C-8 and data needs and a primer on C-8 (and FS-62) toxicology.
Eco Risk Assessment (John G.): Expected timeline and data needs to complete the ecorisk assessment.
Business Risk Assessment (Mike, Rob, Trini): Timelines, plans and data needs to complete the C-8 business risk assessment
WW site issues and data (Roger, Tony, John M.) A summary of WW data that is available on C-8 concentrations in water, C-8 Emissions, C-8 in blood of workers, and a characterization of site concerns.

The first three of these topics will be just for background. Please limit each of these presentations to 15 minutes. The remainder of the first day will be devoted to mapping out a "project plan" for the remainder



of the topics: Please be concise in your preparation but we don't want to limit the time in any way. Rick will be asking you a lot of questions throughout the discussion to enable mapping of the topics. The second day will be spent identifying the coordination areas, developing the synergies, refining the "project plan," and developing a management framework to enable us to meet the plan.

Any questions, please give me a call to discuss.

Thanks,
Rob

Robert F Pinchot 10/22/1999 01:46 PM

To: Richard J Angiullo/AE/DuPont@DuPont, Michael E McCord/AE/DuPont@DuPont
cc: Maurice Astorga/AE/DuPont@DuPont, Gerald L Kennedy/AE/DuPont@DuPont, Gary W Jepson/AE/DuPont@DuPont
Subject: Results from the C-8 Monkey Study

Gerry Kennedy called me last evening and gave me a summary of the results of the monkey study. Since we are not having the meeting today, Rich had asked for a summary of the results. One word of caution on these results: there is one key piece of data that has not been reported yet. Though not likely to change the results, these data are still unknown. We should know next week.

Study Design Summary

6 Months duration with recovery period
Dosing levels of 3, 10, 20/30 mg/kg/day C-8 by gravage. (The study started with the high dose being 30mg/kg/day but the monkeys were not faring too well so they reduced the dose to 20mg/kg/day)
6 monkeys in each group plus 6 control (0 dose)

Key Results

Monkey's reactions

- 4 of the high dose monkeys were in distress
  Liver damage observed
- 1 of the high dose monkeys died
- 1 of the low dose monkeys died
  Unclear cause of death
  No infection or other disease was observed
  Consensus is that the death was C-8 related
- No changes observed in the microscopic pathology of the monkeys

Liver Effects

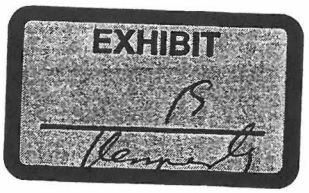
- At all doses liver weight increases were seen
- The response was somewhat dose related but not linear
- There were no histopathic changes in the liver (i.e. the liver cells were not larger)
- Data on liver cell count not back yet (this is the key piece of data missing that I mentioned above)
- Usually only two reasons liver weight increases...either there are more cells or the cells are bigger. If there are more cells, this increases the chance for mutations and could lead increased risk of tumors. No evidence of tumors were found in any of the test monkeys
- It is a strong feeling of the group of toxicologists that were in attendance that C-8 is a very strong liver enzyme inducer (i.e. the liver tries to metabolize it). However, the liver is unable to do anything with it.

Hormonal Effects

- Estrogen and Testosterone: no effects seen
- Some Thyroid changes seen but does not appear to be C-8 related
- Carbohydrate metabolism was OK

Blood Concentrations/Accumulation

- The blood concentrations of the three test groups increased with dose but not linearly as would be expected. For doses of x, 3x, and 7x, equilibrium blood concentrations were approximately y, 1.5y, and 3y, respectively.



- The half life in monkeys is very short. Within a couple of days after dosing stopped, C-8 concentrations in the blood fell to <10% of the peak concentration.
- The material is excreted almost exclusively in the urine as C-8.
- There was no evidence of any accumulation in the tissues of any organ.
- It has been suggested (given this short half-life in monkeys and a seemingly long half-life in humans) that there may be more than one process going on to clear C-8 from the body. Possibly an initial rapid clearing (as seen in monkeys) followed by a very slow clearing mechanism (as seen in humans for which we only have blood concentrations measured years apart). 3M alluded to some studies that they are doing on retirees to look at this issue and said that they would share the results of the studies when complete.

Conclusions

I'd rather not make any firm conclusions until Gerry has a chance to further digest (excuse the pun) the data, the final liver results are in, and other loose ends are tied up. However, I asked Gerry about possible implications for exposure limits. His preliminary opinion is that this data won't allow us to be less conservative (i.e. raise exposure limits). Since we have been very conservative in setting exposure limits for C-8 because of the biopersistence issue, we may not need to be more conservative (i.e. lower exposure limits).

Comment

I took notes over the phone last night and some of my notes are cryptic. I may have mis-interpreted something that Gerry said. Please don't get set in your opinions about this study or C-8 based on this note. Gerry will give a number of us a better appreciation of the results in the meeting next week.

Rob

Monkey Study Presentation by Kennedy

- Ritchey, Pleystis, Wigfield, Zipfel — Parkersburg → Garza
- Kennedy, Paudot, et al (Astuga) — Wilmington

Summary -

Surprises

1) Potency at low dose
2) Differences and to and
3) Cleared from blood more quickly than expected
4) Blood levels plateaued at lower levels
5) Cleared in Urine more quickly

✓ Study - 22 monkeys (Similogolus) 6 controls / 6 mo. exp, 3 mo after feed stop (Recovery)

✓ Study by APME, Covance Lab. (Madison, Wisc lab)

✓ Kennedy pleased with study

✓ 3M oversaw the study

✓ • 1 monkey died on Day 137
1 " " " " 29 (at 20 mg/kg Dose)
• Animals could not tolerate 30 mg/kg

✓ Cells did not appear to be larger - more later

EXHIBIT 20 Kennedy

EID093190

RJZ020460

C-8 Monkey Study

- Goal: A) to determine toxicologic effects of C-8 in the primate following an extended exposure period
  B) to determine potency for producing change (NOEL-LOEL-EL)

- Surprises: - Potency at lower dose (3 mg/kg)
  - Failure of all animals in group to respond similarly
  - Quick plateau of C-8 in blood
    A) Quick clearance from blood
    B) Lack of proportional response (exposure of x, 3x, 6x did not lead to blood concentrations of x, 3x, 6x)

- Subtle vs major toxicity end points

- Hormones unchanged

- Pathology unremarkable, especially in severely affected monkeys

C-8 Monkey Study

Sponsor: APME Ad-Hoc APFO
Toxicology Working Group

Testing Facility: Covance Laboratories
Madison, WI

Study ID: 6329-231

Study Director: Peter Thomford
Study Monitor: Paul Lieder - 3M
Study Representatives: David Farrar - ICI
Reinhart Jung - Clariant
Gerry Kennedy - DuPont
[Giovanni Costa - Mitani]
[George Lin - Daikin]

In Life 9/23/98 - 7/2/99

C-8 Monkey Study

Experimental Details

- Oral dosing - gelatin capsules
- Diet - primate diet, 1 or 2 x/day
  - supplemented fruit/vegetables
- Young adult/adult - 3 ↔ 5 kg

C-8 Monkey Study

Design

Group 1 Control - 6 cynomolgus males
4 6 months
2 6 months & 3 months recovery

Group 2 3 mg/kg - 4 males - 6 months
(1 monkey - died day 137)

Group 3 10 mg/kg - 6 males
4 6 months
2 6 months & 2 months recovery

Group 4 20/30 mg/kg - 6 males
30 - day 1-11
0 - day 12-21
20 - day 22 →6 months
3 monkeys - dosing discontinued Days 43 ↔81
(1 monkey - died day 29)
All sacrificed at 6 months

C-8 Monkey Study

Parameters

I. *In Vivo*

Observed 2x/day
Body weight - weekly
Food consumption - estimate daily
Ocular exams - pre-test/weeks 27 & 40

II. Clinical Pathology

Timing - Pre-test, days 30, 60, 90, 180
Recovery days 30, 60, 90
Hematology - RBC, Hb, PcV, platelets
WBC & diet, reticulocytes, cell-indices
Coagulation - APTT, pro time, fibrinogen
Clin Chem - glucose, UN, creatinine, prot, bilirubin, cholesterol, triglycerides, ALT, AP, AST, GGT, SDH, ions (Ca, etc), amylase, lipase
Urine - standard & urobilinogen, bilirubin

C-8 Monkey Study

III. Blood Hormones
- Timing - 3x pre-dosing, day 30, 60, 90, 180
  Recovery day 30, 60, 90
- Estradiol
  Estrone
  Estriol
  Thyroid stimulate hormone
  Total & free iodothyronine (T3)
  Total & free thyroxin (T4)
  Testosterone
  Cholecystokinin

IV. Exposure Indices
- Serum APFO - 7 days & every 2 weeks thereafter
- Urine APFO - as serum
- Feces APFO - as serum
- Liver APFO - at sacrifice

C-8 Monkey Study

V. Pathology

Complete nescropsy

Organ weights

adrenal, brain, epididymis, kidney,
liver, pancreas, testes, thyroid (parathyroid)

Histopathology (36 tissues)

Additional parameters

- Palmitoyl CoA oxidase
- Cell proliferation
- Bile acid determination

(receptor level determinations)

(bone marrow smear)

C-8 Monkey Study - Results

I. Clinical Observations

Control - none

3 mg/kg - 1 monkey - week 18 - ataxic, hypoactive
no food consumption, limited use
of hind limbs
week 20 - sacrificed (humanely), lost 9.5% bwt (was not going to survive)

3 monkeys - none

10 mg/kg - 6 monkeys - none

30 mg/kg - week 1 - low food consumption
lost 3-7.5% bwt

↓

20 mg/kg - week 3 - 3 monkeys - same as above without marked wt loss, treatment discontinued week 7, 10, 12
1 monkey died week 4
2 monkeys - no clinical signs after week 2

1 - clinical signs at 7 weeks.

All recovered by week 26.

RJZ020468

C-8 Monkey Study - Results

II. Ophthalmology - no findings

III. Body Weight
0/3/10 mg/kg - no differences
20 mg/kg - lower wk 7, 9, 24 (14.3% wt gain of controls)
[30 mg/kg - lower wk 1]

IV. Food Consumption
0/3/10 mg/kg - no differences
20/30 mg/kg - lower (some no food consumption)

V. Blood Hormone
No significant effects (0-30 mg/kg)
Unexplained total thyroxin lower 20 mg/kg
free triiodothyronine lower 20 mg/kg

VI. Clinical Pathology
0/3/10 mg/kg - no differences
30/20 mg/kg - mild ↑ triglycerides
mild ↓ neutrophil, protein, albumin
- 2 distressed - ↑ marked ALT, AST, SDH, (creatine kinase)
↑ mild bile acids
- recovery in off-treatment monkeys

EID093199

RJZ020469

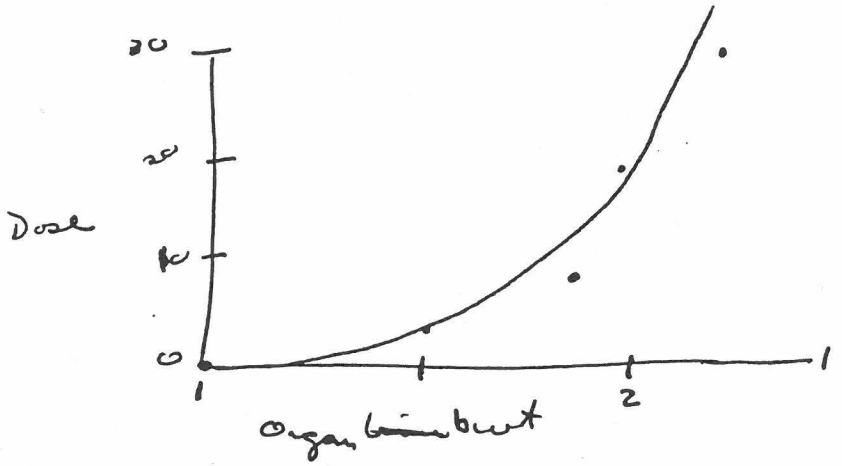
3 mg/kg monkey which died not included in these data

C-8 Monkey Study - Results

VII. Palmitoyl CoA oxidase - expect <4 wks
Cell proliferation - expect <2 wks
Bile acid - no differences

VIII. Pathology
Gross pathology - unremarkable
Organ weight - liver weights elevated
0/3/10/20-30
60/82/83/90 Mean (all test*)
1.5/1.8/1.9/2.4 Organ/bwt (all test*)

Histopathology - unremarkable
Cause of death - unclear in both moribund animals



EID093200

C-8 Monkey Study
Urinary C-8 Levels (ppm)

Group (mg/kg)	Week 2	6	10	14	18	26	28	36	40
0	0/0	0/0.2	0/0	0/0	0/0	0/0	0/0.2	0/0	0/0
3	76/57	69/138	42/35	66/61	67/38	54/39	--	--	--
10	184/430	133/164	182/284	127/129	191/159	114/269	.3/.3	0.05/0.04	0.04/0.04
20/30	949/548	283/369	342	156	177	61	--	--	--

C-8 Monkey Study
Liver C-8 Levels (ppm)

Group (mg/kg)	[Liver]
0	0.04 (0-0.04)
3	5.9 (4.1-6.8)
10	5.9 (3.6-8.7)
20/30	17.2 (8-28)
Recovery	0.8 (0.03-1.2)

C-8 Monkey Study - Conclusions

- Monkeys do not tolerate 20 mg/kg or higher
  - Non-specific response involves liver
  - Recovery complete in off-dosing monkeys
  - Effects include body weight, liver weight
  - No specific histopathological changes
    hormonal changes
- Effects at 10 mg/kg only liver weights
- Effects at 3 mg/kg - 1 death (relationship to treatment?)
  liver weights
- C-8 cleared quickly from urine (proportional to exposure)
- C-8 clears quickly from blood (not proportional)
  - Reaches plateau quickly
  - Leaves system quickly
- C-8 in liver proportional to exposure,
  recovery quick and complete
- C-8 in feces - will get "matrix information"

C-8 Monkey Study - Conclusion

- No observed effect level not attained (<3 mg/kg)
- Potential serious effect in 1/4 monkeys at low dose (liver effect unexplained currently; best explanation could be enzyme induction)
- At AEL/TLV of 0.01 mg/m^3, daily exposure to man is 0.001 mg/kg

C-8 Monkey Study - Issues

- Liver effect (will answer)
- Death of low dose monkey
- Lack of slow elimination as seen in man
  (does human data reflect multi-phase clearance)
- Evaluation of monkey-by-monkey data (in progress)

Thanks for Hanging in there.....

I unexplicably died

EID093206

Privileged and Confidential
Attorney Work Product
Draft
PRELIMINARY REPORT
On

A STUDY OF THE EFFECT OF PERFLUOROOCTANOIC ACID (PFOA) ON RUMEN FERMENTATION

The work reported in this document was conducted in the laboratory of Dr. John Burton, Department of Animal and Poultry Science, University of Guelph, from August 23rd to 31st, 2000.

Procedures:

Two liters of rumen fluid were collected from non-lactating Holstein cows maintained on good quality grass hay. The fluid was collected and handled under CO_2 to maintain anaerobic conditions. Rumen fluid was strained through cheesecloth to remove large particulate matter then 50 ml immediately pipetted into 125 ml glass volumetric flasks, which contained 2 g of ground alfalfa leaf meal. The flasks were placed in a water bath maintained at approximately 39 °C . Once the temperature had equilibrated (within 1 hour) 1 ml of distilled water containing PFOA was added to flasks allocated to either low or high treatments to give final PFOA concentrations of either 0.2 ppm or 200 ppm respectively. Flasks were removed in triplicate from the bath and measured for pH and microbial numbers at 12, 24, 48 and 72 hours of incubation. Thus a total of 12 flasks per treatment, including controls (no PFOA added), were used. No buffers apart from those naturally occurring in the rumen fluid were used in this trial.

Sample pH was obtained using a Corning model 115 pH meter. Readings were taken immediately upon removing the flask from the water bath. Microbial counts were obtained using a hemocytometer having a chamber volume of 0.1 cu mm and a Zeiss light microscope. Duplicate counts were made on each flask. Flasks were maintained in a heated water bath until counting was completed. The order of counting the flasks was randomized.

Results:

As the data in Table 1 indicate, with very little buffering capacity in the rumen fluid the pH decreased rapidly within the first 12 hours of incubation. The change in pH was relatively slow from 12 to 72 hours although it did continue to decrease. Treatment with PFOA had no significant effect on the pH of the samples, nor the apparent rate at which pH changed.

In contrast, the PFOA appears to have considerable effect on microbial survival (see Table 2.). By 12 hours of incubation the visible microbes in the PFOA treated flasks had decreased to approximately 50 % of the numbers visible in the control flasks. There was little difference in numbers between concentrations of PFOA at this time. By 24 hours there appears to be little change in numbers in the control flasks. Numbers in the

lower concentration of PFOA appear to recover slightly, however this change is not statistically significant. The microbes in the higher concentration of PFOA change very little from 12 to 24 hours and are significantly lower than the control data. All microbial activity had ceased by 48 hours of incubation, probably because the available substrate had been depleted. No 0 time counts were made on the rumen fluid.

Table 1. The effect of PFOA on pH of rumen samples					
Treatment	Sample Time (hr)				
	0	`12	24	48	72
Control	6.80	5.85	5.60	5.45	5.35
		5.90	5.70	5.35	5.40
		5.80	5.60	5.40	5.40
Mean ± sd		5.85 ± 0.05	5.63 ± 0.06	5.40 ± 0.05	5.38 ± 0..03
Low	6.80	5.80	5.70	5.40	5.40
		5.80	5.65	5.40	5.40
		5.70	5.70	5.45	5.40
Mean ± sd		5.76 ± 0.06	5.68 ± 0.03	5.42 ± 0.03	5.40
High	6.80	5.80	5.65	5.50	5.40
		5.80	5.70	5.35	5.45
		5.75	5.80	5.45	5.40
Mean ± sd		5.78 ± 0.03	5.72 ± 0.08	5.43 ± 0.08	5.42 ± 0.03

Low: 0.2 ppm PFOA; high: 200 ppm PFOA

Table 2. The effect of PFOA on rumen microbial numbers in samples of rumen fluid (includes both bacteria and protozoa)					
Treatment	Sample Time (hr)				
	0	`12	24	48*	72*
Control	No count	22	18		
		16	18		
		14	18		
Mean ± sd		17.3 ± 4.2	18		
Low	No count	11	10		
		5	10		
		12	17		
Mean ± sd		9.3 ± 3.8	12.3 ± 4.0		
High	No count	10	10		
		6	8		
		10	5		
Mean ± sd		8.6 ± 2.3	7.6 ± 2.5		

Low: 0.2 ppm PFOA; high: 200 ppm PFOA

* No living microbes were visible at 48 and 72 hours in any samples from any of the treatments.

Report prepared by:
Dr. John H. Burton
Professor, Animal Nutrition, Department of Animal and Poultry Science
University of Guelph

LE 1740

Leigh Belcher
Written by GLK 5-25-99

DRAFT

AMMONIUM PERFLUOROOCTANOATE
ACCEPTABLE DAILY INTAKE-COMMUNITY

The toxicity data base regarding the biological effects of ammonium perfluorooctanoate (CAS 3825-26-1;C-8 or APFO) has been reviewed for the purpose of estimating a daily human dose which would be expected to be without adverse effect on human health. The suggested value is 6 micrograms per individual per day. The following gives the background information and the thought process used to arrive at the above number.

Data Base:

C-8 has a moderate acute oral toxicity with an LD50 in rats of 470 mg/kg (1). Dermal application of 1,500 mg/kg to the skin of rats produced clinical signs of response including weight loss and labored breathing (2).The material is mildly to moderately irritating to rabbit skin (3) and instillation of the solid material into the rabbit eye produced moderate irritation which resolved slowly (4). Acute inhalation shows the material to be moderatley toxic with a 4 hour LC50 in rats of 800 mg/m3 (5).

Repeated exposure toxicity studies have demonstrated that the liver is the target of C-8 toxicity. In a 2 week inhalation study in which rats were exposed for 6 hours per day, 5 days per week to either 11 or 83 mg/m3 C-8, liver degeneration, enlargement, and increases in serum levels of liver enzymes were seen with the effects more pronounced at the higher level (6). To titrate these effects and confirm the findings, a second study using the same design was conducted at exposure levels of 1, 7.6, and 83.9 mg/m3. In this experiment, some of the rats exposed at 83.9 mg/m3 died and liver changes as seen earlier occurred at both 7.6 and 83.9 mg/m3. The liver changes reversed as the rats were allowed a recovery period. The no-observed-adverse-effects (NOAEL) level in this study was 1 mg/m3 (7). Repeated exposure studies with C-8 following both oral (8) and dermal (9) exposures confirm that the liver is the target organ in the rodent. A study reported in the monkey following oral treatment is not acceptable for use in this analysis since the material was not well tolerated orally as a part of the population showed emesis throughout the experiment. Thus the actual doses delivered to the animals was not well characterized (8). A 6-month oral study in monkeys is currently underway and the confirmed results should be available for use in this assessment 4Q/99.

2 wks

A lifetime feeding study in rats has shown C-8 to produce liver toxicity following feeding of either 30 or 300 ppm (10). At 300 ppm (but not 30 ppm), an increase in testicular Leydig cell adenomas were seen and a marginal increase in testicular tumors was observed. In a follow-up study in which 300 ppm was fed to rats for 2 years to look at the mechanisms of C-8 induced changes, increases in hepatic adenomas, Leydig cell adenomas, and pancreatic acinar cell adenomas were seen (11). The mechanism of these changes involved peroxisome proliferation and dosage-related hormonal changes which both show a threshold for effect.

incidence of tumors low
profiles consistent with geriatric rats

EXHIBIT 28
Kennedy

LAB000047

EID108184

C-8 does not appear to be mutagenic with negative results seen in the Ames Salmonella assay and in Saccharomyces cerevisiae (8).

The developmental toxicity of C-8 has been examined in rats following either oral (12) or inhalation (13) exposures and no signs of enhanced fetal sensitivity was seen with no structural malformations associated with C-8 treatment found. Pregnant rabbits given oral doses of C-8 again showed no fetal effects (14).

A suggested relationship between C-8 exposure in man and prostate cancer (15) has been rejected following more careful examination of the exposed population (16-G.Olson,1998 or 9). Examination of the work force potentially exposed to C-8 in manufacturing operations showed no change in serum enzyme levels used as diagnostic for liver damage (18-Fayerweather).

In man, C-8 has a long half-life in human blood. A study of occupationally exposed workers producing the material showed organic fluoride levels ranging from 1 to 71 ppm with an individual having a value of 70 ppm being removed from further exposure and showing 39 ppm 18 months later (19-Ubel).
The slow removal of C-8 from human blood has been the hallmark concern in human risk assessment. Sex and species differences in clearance have been demonstrated but, although there is little data from human females, it appears as if both sexes show slow clearance of C-8 from the blood.

Control levels/Acceptable exposures:

Based on the 1 mg/m3 NOAEL determined in rats inhalaling C-8 daily for 2 weeks, a workplace concentration of 0.01 mg/m3 (8 hour TWA) was recommended. The dose causing minimal liver damage to rats in a 2-year feeding study was 30 ppm or 1.5 mg/kg/day. Assuming total absorption of an airborne dose, a 70 kg person breathing 10 m3 per workshift would , at 1.5 mg/kg, be exposed to 10.5 mg/m3—considerably higher (1000x) than the recommended workplace control limit. Since the community would be receiving C-8 24-hours per day (rather than 8 hour exposed, 16 hour non-exposed as in the workplace) and because the community would include those which could be more sensitive to the effects of C-8 (the young, aged, infirmed), a reduction in the airborne limit for the general community to 0.0003 mg/m3 was recommended. Overriding the quantitative aspects here is the knowledge that C-8 persists in the blood for extended periods.

At the 0.0003 mg/m3 limit, it is expected that man can be exposed daily without adverse health effects. Since the amount of air breathed per 24 hours is approximately 20 m3, a daily exposure to 6 micrograms (0.0003 mg/m3 X 20m3) would be expected to be without health consequences. This amount of chemical could be allowed to enter the body on a daily basis.

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What Are The Issues Associated with Development of Remediation Screening Levels for C-8?

ISSUE	OPTIONS
Toxicity Criterion to be Applied:	*Review and interpret open literature *Use "AEL-Based" Allowable Daily Intake of 100 µg/d(1) *Use "CEG-Based" Allowable Daily Intake of 6 µg/d(2) *Postpone development until primate study is completed
Exposure Assumptions to be Used:	*Assume coincident exposure of on-site workers to multiple media(3) *Assume that workers are discretely exposed to individual media(4)

(1) AEL is based on a two-week inhalation study NOEL of 1 mg/m3.
(2) Based on AEL divided by 30 to account for 8-hr/day vs. 24-hr/day exposure, and exposure of sensitive individuals.
(3) Confounded by the fact that the degree of exposure to the various media is unknown.
(4) This is the tact followed by EPA Region III in the development of their RBCs.

Prepared at Request of Counsel

URS Greiner Woodward Clyde

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DATE: 4-30-99

PAGE 1 OF: 4

TO: Tim Bingman	Andrew Hartten	FROM: Pat Whitehead	
FIRM: DuPont	DuPont	SUBJECT:	
FAX NO: 412-749-5246	302-892-7643	CC:	

MEMO:

For discussion

EXHIBIT
29
Kennedy

EID103032

ASI1010179

Pat Westphal
Washington Works
4-30-99

1. AEL = 0.01 mg/m³ is protective of workers
   - 100 x lower than NOEL (rats) of 1 mg/m³ [2-week inhalation study]
   - 1000 x lower than dose associated with marginal effect level for liver toxicity and NOEL for tumors (2-yr rat feeding study)
   - dose estimate assumes 100% absorption in lung and 10 m³/day inhalation (Not conservative)
   - AEL derivation is independent of exposure time and duration

2. $CEG_{air} = \frac{AEL}{\text{safety factor}} = \frac{0.01\ mg/m^3}{30} = 0.3\ ug/m^3$

Community Exposure Guideline
- safety factor of 3 for 8 vs 24 hr/day exposure
- safety factor of 10 for population diversity
- note the safety factor is partly based on exposure time, which is not an assumption in the AEL.

3. $CEG_{water}\ (ug/L) = \frac{20\%\ \text{of intake by air}\ (ug/day)}{2\ L\ water/day}$

$= \frac{0.2}{2\ L/day} \times CEG_{air}\ (ug/m^3) \times 20\ m^3/day$

$= \frac{0.2}{2} \times 0.3 \times 20 = 0.6\ ug/L$ Day 1 ug/L

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H. Target level (TL) for soil (worker exposure).

Alt 1:
- Assume background air = AEL
- Assume 10% increase in intake is allowable
- Assume ½ of the 10% is from soil (rest from water)
- Intake from air = $\frac{0.01\ mg}{m^3} \times \frac{10.5\ m^3}{day} = 0.100\ mg/day$
- Allowable intake from soil = $\frac{0.100\ mg}{d} \times .05 = \frac{0.005\ mg}{d}$
- TL soil (ingestion route) = $\frac{0.005\ mg}{d} \times \frac{1\ day}{100\ mg\ soil} \times \frac{10^6\ mg}{kg}$

$= \frac{50\ mg}{kg}$

- TL soil (ing + dermal) = $\frac{25\ mg}{kg}$ assuming dermal intake approximately = soil intake

Notes: Inhalation from wind erosion of soil is negligible.
Max. observed RFI conc. = 48 mg/kg.

Features of TL derivation:
a. extremely conservative (assumes exposure to AEL as background condition)
b. intake calculations assume 100% absorption in lung
c. Must make assumptions re dermal absorption
d. Could be too conservative
e. Avoids deriving an RFD.

Alt 2
- Assume background air < AEL
- Assume allowable total intake = 0.100 mg/day

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5. TL for groundwater (workers)

- $TL = 0.005 \frac{mg}{day} \times \frac{1\ day}{1\ L} = 5 \frac{ug}{L}$

  Allowable intake
- Based on same assumptions as TL_{soil} alternative 1

6. TL for groundwater (fenceline resident)

- Assume soil and air exposure pathways are incomplete (groundwater is only exposure medium)
- Assume allowable intake = $CEG_{air} \times \frac{20\ m^3}{day}$

$$= \frac{0.3\ ug}{m^3} \times \frac{20\ m^3}{d}$$

$$= 6\ ug/day$$

- TL water (ingestion) = $\frac{6\ ug}{day} \times \frac{1\ day}{2\ L} = 3 \frac{ug}{L}$
- TL water (ing + dermal) = 1.5 ug/L
- Use MW data?

7. Construction workers (soil)

- Potential exposure / intake from soil at TL = 25 or 50 ppm could exceed allowable level based on AEL, because soil ingestion rate would be higher, and inhalation of PM released from soil might contribute to overall intake.
- (Hot spot in RBL).

8. Construction workers (groundwater)

- Minimal ingestion, possible dermal contact
- Use MW data?

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